

**A CLINICAL STUDY AND MANAGEMENT OF GASTRIC OUTLET  
OBSTRUCTION IN ADULTS**



**By**

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I hereby declare that this dissertation **“A CLINICAL STUDY AND MANAGEMENT OF GASTRIC OUTLET OBSTRUCTION IN ADULTS”** is a bonafide and genuine research work carried out by me under the guidance of PROF.P. RAGUMANI., M S., Professor, Department of General Surgery, MMC & RGGGH, CHENNAI.

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**“A CLINICAL STUDY AND MANAGEMENT OF GASTRIC OUTLET**  
**OBSTRUCTION IN ADULTS”**

**is a bonafide**

**Research work done by Dr. G PRABHU in partial**  
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## **ABSTRACT**

Gastric Outlet Obstruction implies complete or incomplete obstruction of the distal stomach, pylorus or proximal duodenum. Gastric outlet obstruction is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying.

Now in the era of H2 blockers and proton pump inhibitors, incidence of duodenal ulcer producing gastric outlet obstruction has been decreasing as symptomatic ulcer begin to respond to medical treatment, and at the same time the incidence of antral carcinoma of stomach producing gastric outlet obstruction has comparatively increased which may be due to increased early diagnosis of the condition with the help of flexible fibre optic endoscope.

### **Aims and objectives:**

1. To determine the relative incidence of benign and malignant gastric outlet obstruction.
2. To study the modes of presentation of gastric outlet obstruction.
3. To study the outcome of management of gastric outlet obstruction.

### **Methodology:**

In this study, 50 in-patients presenting with features of gastric outlet obstruction to Rajiv Gandhi Govt. General Hospital from October 2013 to September 2014 have been studied.

An elaborate study of these cases with regard to the history, clinical features, routine and special investigations, pre-operative treatment, operative findings, postoperative management and complications in post-operative period is done.

### **Results and Observations:**

Of the 50 cases of gastric outlet obstruction 34 had carcinoma antrum (72%) 14 had cicatrized duodenal ulcer (28%) and 1 had gastric outlet obstruction secondary to corrosive ingestion.

The age incidence of the patients in this study ranged from 29-76 years with a mean of 53.32years. In case of obstruction secondary to duodenal ulcer the maximum age incidence is between 40-49years. The maximum age incidence of gastric outlet obstruction due to carcinoma antrum is 50-59years. In this series, 36 patients (72%) were males and 14 patients (28%) were female. Male to female ratio (M:F) is 2.57:1. M:F ratio in cicatrized duodenal ulcer is 2.5:1 and in carcinoma antrum is 2.4:1. 52% of the patients were manual labourers who gave a history of irregular diet habits. 68% of patients had history of smoking and 66% had history of alcohol intake. Post – prandial vomiting and epigastric pain are the main symptoms (96%) in this series. Other symptoms included anorexia (84%), weight loss (72%), post prandial epigastric fullness (68%), haematemesis (24%), malena (64%) and constipation (48%). Pallor was present in 68% and dehydration in 62%. Blood group ‘O’ was common in



cicatrized duodenal ulcer patients (57.14%) whereas blood group 'A' was common in malignant cases (58.82%).

### **Conclusion:**

Number of cases with cicatrized duodenal ulcer as the chief etiological factor for gastric outlet obstruction is diminishing and the number of cases of antral carcinoma of stomach as the cause of gastric outlet obstruction is increasing. Upper Gastro intestinal endoscopy should be mandatory in all suspected cases of gastric outlet obstruction. It can diagnose the cause of obstruction very effectively than any other investigative modality. Effective treatment in carcinoma stomach depends on early diagnosis.

### **LIST OF ABBREVIATIONS**

5-FU	-- 5-Fluoro-Uracil
APD	-- Acid Peptic Disease
ECF	-- Extra Cellular fluid
GD	-- Gastro Duodenal
GEJ	-- Gastro Esophageal Junction
GI	-- Gastro Intestinal
GJ	-- Gastro Jejunostomy
GOO	-- Gastric Outlet Obstruction
GFR	-- Glomerular Filtration Rate
H2RA	-- Histamine 2 Receptor Antagonist

HCl	-- Hydro Chloric Acid
HSV	-- Highly Selective Vagotomy
MAO	-- Maximum Acid Output
MEN	-- Multiple Endocrine Neoplasia
NHL	-- Non-Hodgin's Lymphoma
NSAID	-- Non-Steroidal Anti Inflammatory Drugs
OGD scope	-- Oesophago-Gastro-Duodenoscope
PPI	-- Proton Pump Inhibitor
PUD	-- Peptic Ulcer Disease
SMA	-- Superior Mesenteric Artery
VGP	-- Visible Gastric peristalsis.

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## INTRODUCTION

Gastric Outlet Obstruction implies complete or incomplete obstruction of the distal stomach, pylorus or proximal duodenum. This may occur as an obstructing mass lesion, external compression or as a result of obstruction from acute enema, chronic scarring and fibrosis or a combination of both.

Gastric outlet obstruction was described by Sir James Walton as “The stomach you can hear, the stomach you can feel and the stomach you can see”.

Gastric outlet obstruction is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying.

Gastric Outlet Obstruction may be caused by a heterogeneous group of diseases that include both benign and malignant conditions. In adults, mechanical obstruction due to ulcers, tumours or gastric polyps are common causes of gastric outlet obstruction.

Until introduction of effective ulcer therapy duodenal ulcer was the commonest cause of gastric outlet obstruction and malignancy was attributed to only 20% of the cases. But, now in the era of H<sub>2</sub> blockers and proton pump inhibitors, incidence of duodenal ulcer has been decreasing as symptomatic ulcer begin to respond to medical treatment, although this has not reflected to changes of complication like bleeding and perforation. At the same time the incidence of antral carcinoma of stomach producing gastric outlet obstruction

has comparatively increased, which may be due to increased early diagnosis of the condition with the help of flexible fibre optic endoscope.

This study has been taken up to review the changes in presentation of gastric outlet obstruction in view of changing trends in the management because of new drugs and investigatory modalities. The lack of uniformity in criteria in accepting a case of gastric outlet obstruction lead to differences in incidences and clinical features in different centres, still, any one of the following can be used to diagnose gastric outlet obstruction.

- Projectile vomiting of undigested food consumed previous day.
- Palpable hypertrophied stomach.
- Visible gastric peristalsis.
- Gastric succussion splash 3-4 hours after the last meal.
- OGD scopy.
- Demonstration at operation of grossly narrowed gastric outlet.

In managing gastric outlet obstruction, measures employed are designed to

- Improve the local condition of stomach
- Correct fluid and electrolyte imbalance.
- Correct anaemia, hypoproteinemia and vitamin deficiency.
- Treatment of etiological conditions.

## **AIM AND OBJECTIVE OF THE STUDY**

1. To determine the relative incidence of benign and malignant gastric outlet obstruction.
2. To study the modes of presentation of gastric outlet obstruction.
3. To study the outcome of management of gastric outlet obstruction.

## **REVIEW OF LITERATURE**

In a study by Misra SP, Dwivedi M, Misra V, from M. L. N. Medical College, Allahabad, India in 1998, Seventy-four patients with gastric outlet obstruction underwent upper gastrointestinal endoscopy and biopsy specimens were obtained. In 56 patients (76%) the cause of the gastric outlet obstruction was malignant. Even in a developing country like India, malignancy is the commonest cause of gastric outlet obstruction and endoscopic biopsy specimens should be obtained in all patients with gastric outlet obstruction.

In a study by Shone DN, Nikoomanesh P, Smith-Meek MM, Bender JS, of 33 patients with gastric outlet obstruction admitted to Johns Hopkins Bayview Medical Centre, Baltimore, Maryland, USA between July 1990 and November 1993, 61% (20 patients) had malignancy as the cause of their gastric outlet obstruction. 39% (13 patients) had benign disease. They concluded that the incidence of malignancy in patients presenting with gastric outlet obstruction is greater than 50% and the aetiology of gastric outlet obstruction cannot be predicted by age, history of peptic ulcer disease, or nonsteroidal anti-

inflammatory drug use. The endoscopic treatment of gastric outlet obstruction should be approached with caution because malignancy cannot be reliably excluded by endoscopic or radiological studies.

A study by Ann Roy, Micheline Kim, John Christein, and Shyam Varadarajulu, included 29 patients who underwent stenting and 75 who underwent surgical GJ. Both modalities were technically successful and relieved gastric outlet obstruction in all cases. But compared with surgical GJ, the median post-procedure length of stay was significantly lower for enteral stenting (1.5 vs. 10.7 days,  $p < 0.0001$ ) and there was no difference in rates of delayed complications between stenting and surgical GJ (13.8 % vs. 6.7 %;  $p = 0.26$ ). The technical and clinical outcomes of surgical GJ and endoscopic stenting appear comparable, but stent placement is less costly and is associated with shorter length of hospital stay.

In a retrospective review by Zhang LP, Tabrizian P, Nguyen S, Telem D, Divino C of Twenty-eight patients (16 had malignancy, 7 had PUD, 3 had Crohn's disease, and one had obstruction of unclear cause) who underwent Laparoscopic gastrojejunostomy (LGJ) at Mount Sinai Medical Centre, New York from 2004 to 2008. Patients regained bowel function at a median of 3 days and remained in the hospital for a median of 8 days. There were 4 major postoperative complications (14%): 1 anastomotic leak and 1 trocar-site haemorrhage requiring reoperation and 2 gastrointestinal bleeds requiring endoscopic intervention. There were 5 minor complications (18%), including a

partial small bowel obstruction, 1 patient developed bacteraemia, and 3 patients had delayed gastric emptying. One patient had persistent gastric outlet obstruction requiring reoperation 3 months later. Laparoscopic gastrojejunostomy can be performed for gastric outlet obstruction with improved outcome and an acceptable complication rate compared to the open gastrojejunostomy reported in the literature.

A study of 15 consecutive patients, aged 29 to 75 years who underwent laparoscopic truncal vagotomy and gastrojejunostomy for gastric outlet obstruction between 1996 and 2000 was done by Siu WT, Tang CN, Law BK, Chau CH, Yau KK, Yang GP, Li MK. In Pamela Youde Nethersole Eastern Hospital, Chai Wan, Hong Kong, SAR & China. The mean operative time was 114 minutes. Eleven patients were discharged by postoperative day 10; the remaining 4 patients had delayed gastric emptying which settled with conservative treatment. With an average follow-up period of 80 months, patients were classified as Visick I (n = 7), II (n = 5), III (n = 1), and IV (n = 2). Laparoscopic truncal vagotomy and gastrojejunostomy is technically feasible for patients with benign gastric outlet obstruction and is associated with satisfactory perioperative and long-term outcome.

A comparative study to know the outcome of stent placement and palliative open gastrojejunostomy for GOO secondary to gastric cancer was done by Maetani I, Akatsuka S, Ikeda M, Tada T, Ukita T, Nakamura Y, Nagao J, Sakai Y in Ohashi Medical Centre, Toho University, Ohashi, Tokyo, Japan



between September 1994 and September 2004. Twenty-two patients underwent palliative enteral stenting, and 22 patients were subjected to surgical gastrojejunostomy (bypass). An improvement in performance score after the procedure was observed in both groups (stent group;  $P=0.0264$ ; bypass group;  $P=0.0235$ ). Minor complications were observed in 1 patient in the stent group and in 4 in the bypass group. No mortality or severe complications were observed for either group. Self-expandable metallic stent placement is a safe and efficacious procedure for palliation, with shorter operating time (30 vs. 118 min;  $P<0.0001$ ) and more prompt restoration of oral intake (2 days vs. 8 days;  $P<0.0001$ ) compared to surgical alternatives in patients with GOO caused by gastric cancer.

In a study by Hala Mansoor and Muhammed Aasim Yusuf in 69 patients who had documented gastric outlet obstruction and underwent endoscopic stenting from August 2008 till January 2012 was reviewed retrospectively at Shaukat Khanum Memorial Cancer Hospital & Research Centre, Lahore, Pakistan, 85.5% and 80% benefited from stent insertion within 7 and 28 days after stent placement respectively and Stent related adverse events occurred in 10 patients (14%), including stent blockade in 7 and stent migration in 3 patients. Endoscopic enteral stenting promptly increases oral intake in the majority of patients with malignant gastric outlet obstruction and is a safe procedure with a low rate of serious complications.

## **EMBRYOLOGY AND ANATOMY**

### **Development of the stomach and Duodenum:-**

The stomach, along with the first and second parts of the duodenum, is derived from the foregut. Thus the stomach and proximal duodenum are supplied by branches of the Celiac artery and the distal duodenum by branches of the superior mesenteric artery.

The stomach can be recognized as a fusiform dilatation at the end of the fourth week in the 4 mm. embryo. At the 10 mm, stage the characteristic curvatures of the stomach are readily discernible. The stomach undergoes differential growth resulting in a considerable change in its shape and orientation. The original ventral border comes to face upwards and to the right and becomes the lesser curvature. The dorsal border now points downwards and to the left and becomes the greater curvature. The original left surface becomes anterior and the original right surface becomes posterior. The displacement and the rotation of the stomach have been variously attributed to its own growth changes, extension of the pancreaticoenteric recess and pressure by the rapidly growing liver.

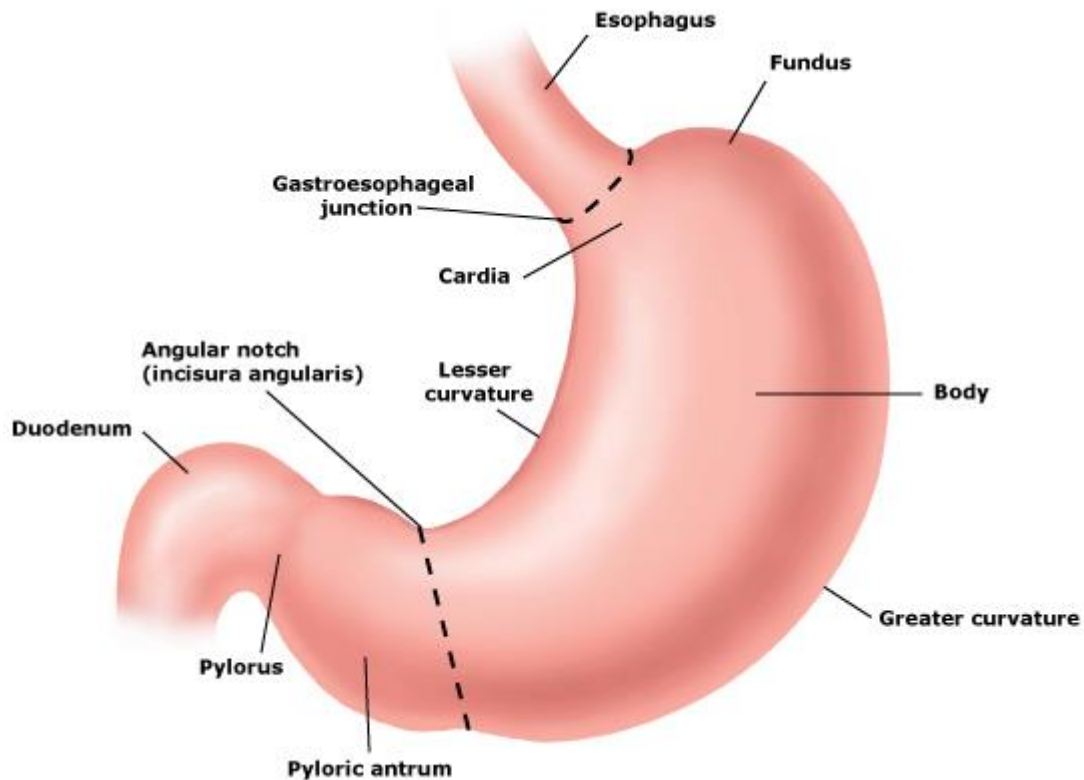
The part of the gut that gives rise to the duodenum forms a loop attached to the posterior abdominal wall by a mesentery (mesoduodenum). Later this loop falls to the right. The mesoduodenum fuses with the peritoneum of the posterior abdominal wall so that most of the duodenum becomes retroperitoneal.

## ANATOMY

The stomach is a muscular bag fixed at both ends and subject to great variation in size in conformity with the volume of its contents. It is the most dilated part of the alimentary canal and is situated between the end of the oesophagus and the duodenum. It lies in the epigastric, umbilical and the left hypochondriac regions of the abdomen. Its shape and position are modified by changes within itself and the surrounding viscera and no one form or position is typical. Its mean capacity varies with age, being about 30 ml at birth, increasing gradually to about 1 litre at puberty and commonly reaching up to about 1.5 litres in the adult.

The stomach is grossly divided into 5 regions

1. The cardia is the portion of the stomach immediately adjacent to the cardiac orifice.
2. The fundus is the upper convex dome situated above the level of the cardiac orifice. It is commonly distended with gas which is seen clearly in X-ray films under the left dome of diaphragm.
3. The body of the stomach extends from the fundus to a vertical line drawn from the incisura angularis.
4. The pyloric antrum extends from the body to the sulcus intermedius.
5. The pyloric canal extends from the sulcus intermedius up to the pyloric opening which is identified on the surface by the pyloric vein of Mayo.

**FIGURE 1. GROSS ANATOMY OF STOMACH**

The stomach has 2 curvatures, the lesser curvature and the greater curvature. The lesser curvature is concave and forms the right border of the stomach. It provides attachment to the lesser omentum. The most dependent part of the curvature presents the angular notch or incisura angularis. The greater curvature is convex and forms the left border of the stomach. It provides attachments to the greater omentum, gastrosplenic and gastrophrenic ligaments.

The stomach is covered by the peritoneum all around except for a small triangular area on the posterior surface close to the cardiac orifice. Anteriorly, the stomach is related to the liver on the right side, and on the left, the diaphragm separates it from the base of the left lung, left pleura, pericardium,

the 6th to 9th ribs and corresponding intercostal spaces. Posteriorly the lesser sac separates it from the diaphragm, left suprarenal gland, left kidney, splenic artery, pancreas, transverse mesocolon and splenic flexure of colon. The greater sac separates the stomach from the spleen.

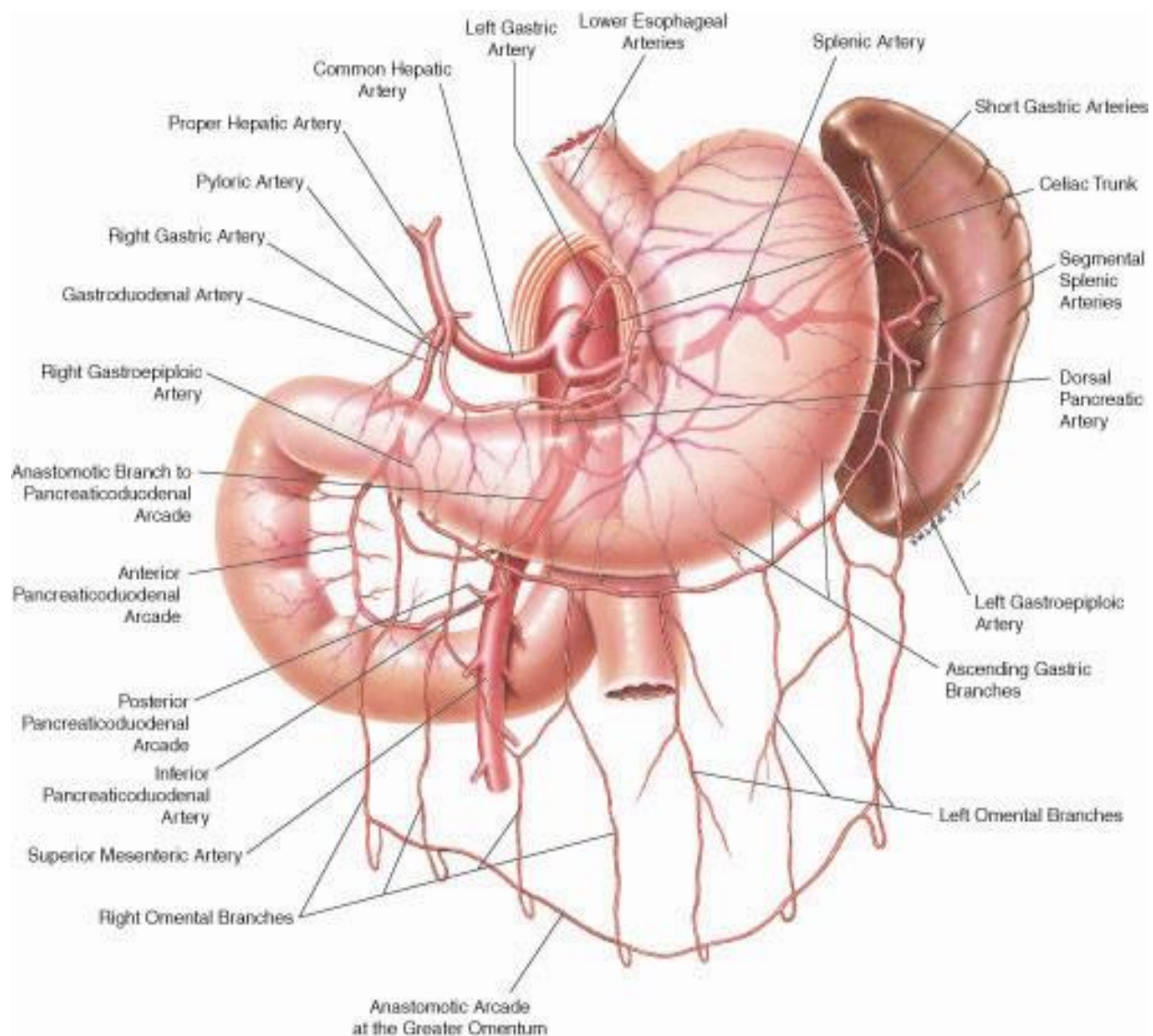
Duodenum is the first part of small intestine and is about 10 inches (25 cms) long. It makes a C-shaped loop around the head of the pancreas and lies at the level of L1-L3. It is divided into 4 parts.

### **Blood Supply of Stomach**

The stomach has a blood supply so extensive and interconnected that 3 of the 4 major nutrient arteries can be ligated without causing necrosis or significant dysfunction. The stomach receives its blood supply through its two mesenteric borders by the left gastric (from the celiac axis), the right gastric and the right gastro-epiploic arteries (from the common hepatic artery) and the left gastro-epiploic and short gastric arteries (from the splenic artery). The right and left gastric arteries run in the lesser omentum adjacent to the lesser curvature while the right and left gastro-epiploic arteries and vasa brevia run within the greater omentum adjacent to the greater curvature. These arteries supply the stomach by sending off specific anterior and posterior gastric branches that penetrate the stomach's muscular coat close to the lesser and greater curvature. On reaching the submucosa, these branches ramify extensively throughout the entire submucosa. Maintaining a relatively larger calibre, these submucosal ramifications anastomose frequently with each other to form the sub-mucosal

plexus which consists of both arteries and their venous counterparts. Independent branches from the submucosal plexus supply the mucosa everywhere except in the lesser curvature which receives branches directly from the right and left gastric arteries. The gastric veins commence as straight vessels between the mucosal glands and these drain into the sub-mucosal veins. They then accompany their corresponding arteries to ultimately drain into the splenic and superior mesenteric veins.

**FIGURE 2 THE BLOOD SUPPLY OF STOMACH.**



## **Nerves supply of stomach**

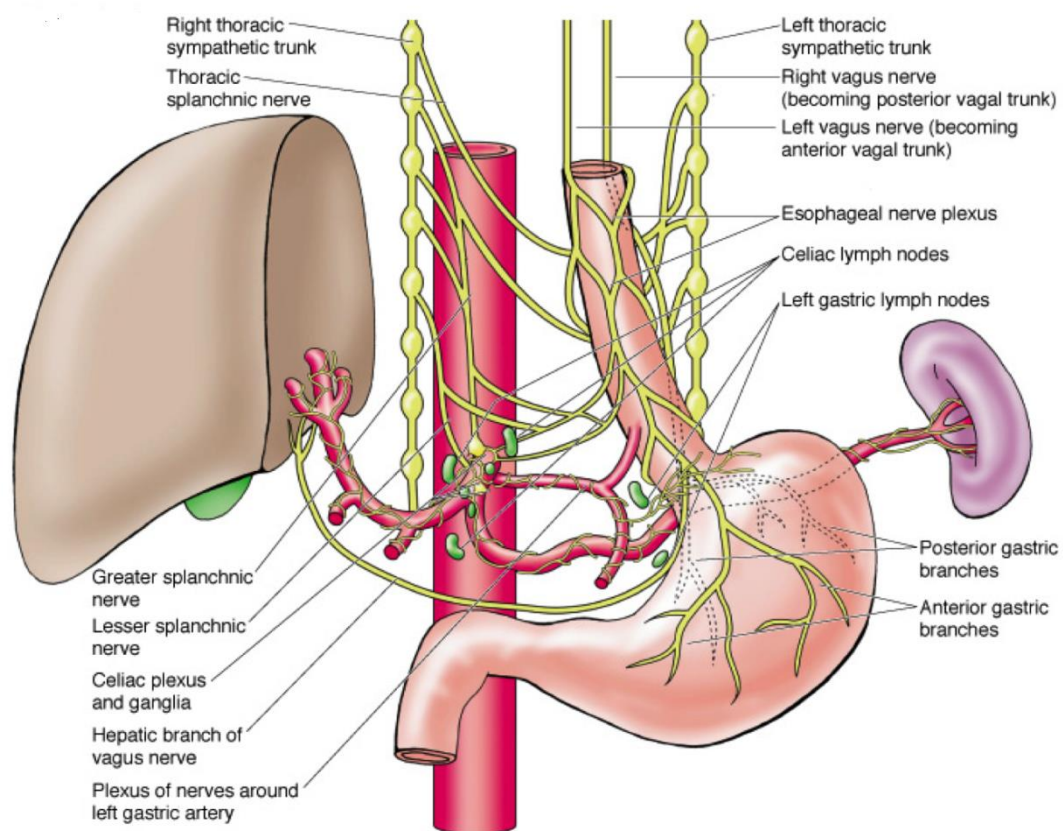
The sympathetic supply is from the celiac plexuses. Pre-ganglionic efferent fibres destined to the stomach and duodenum leave the spinal cord (fifth or sixth to ninth or tenth thoracic segments) traverse their respective sympathetic ganglia without synapse and unite to join the greater splanchnic nerves. On reaching the celiac ganglia the pre-ganglionic fibres form a synapse and unite to join the greater splanchnic nerves. On reaching the celiac ganglia the preganglionic fibres form a synapse with the postganglionic fibres that go to the stomach and proximal duodenum by way of the various branches of the celiac artery. The afferent system consists of a single neuron that returns along the same pathways. The afferent sympathetic fibres carry the pain fibres.

The parasympathetic supply is derived from the vagus. It is the secretomotor nerve to the stomach. The anterior vagus has 3 branches: (1) The hepatic branch (2) The pyloric nerve of McCrea and (3) nerve of Latarjet or the anterior gastric branch (Fig. 2).

The pyloric nerve runs within the lesser omentum to the distal antrum and pylorus midway between the hepatic and anterior gastric branches. But the pyloric branch is more often absent than present. When absent it may be incorporated with the hepatic or the anterior gastric branch. The anterior gastric branch runs along the lesser curvature within the lesser omentum to reach the pylorus. The terminal portion of the nerve resembles the crow's foot.

The posterior vagus gives rise to 2 branches namely (1) the posterior gastric branch (posterior nerve of Latarjet) and (2) the celiac branch. The posterior gastric branch is shorter and has fewer branches than that of the anterior nerve and it usually ends in the antrum. The celiac branch is the largest of the vagi branches and it always descends within the pancreatico duodenal fold as one large fibre to the celiac and the superior mesenteric autonomic plexuses.

The branches to the proximal fundus usually arise from the nerves of Latarjet. But sometimes it may arise higher up from the oesophageal plexus above the hiatus and is termed as Grassi's criminal nerve.



**FIGURE 3. THE NERVE SUPPLY OF STOMACH.**



## **Lymphatic Drainage**

The lymph channels follow the artery. The lymph originates primarily in the mucosa and drains into the lymphatic sub-mucosal plexus which is as rich and extensive as that of the arterial and venous sub-mucosal plexus. They also anastomose with the sub-mucosal plexus of the oesophagus while the duodenum is relatively devoid of sub-mucosal plexus and therefore sub-mucosal spread of carcinoma into the duodenum is unusual. The sub-mucosal plexus then drains into the sub-serosal plexus just beneath the peritoneum. From this point the lymphatic drainage continues through extrinsic channels which are divided into 4 sets which correspond to the 4 zones of arterial supply (Fig. 3)

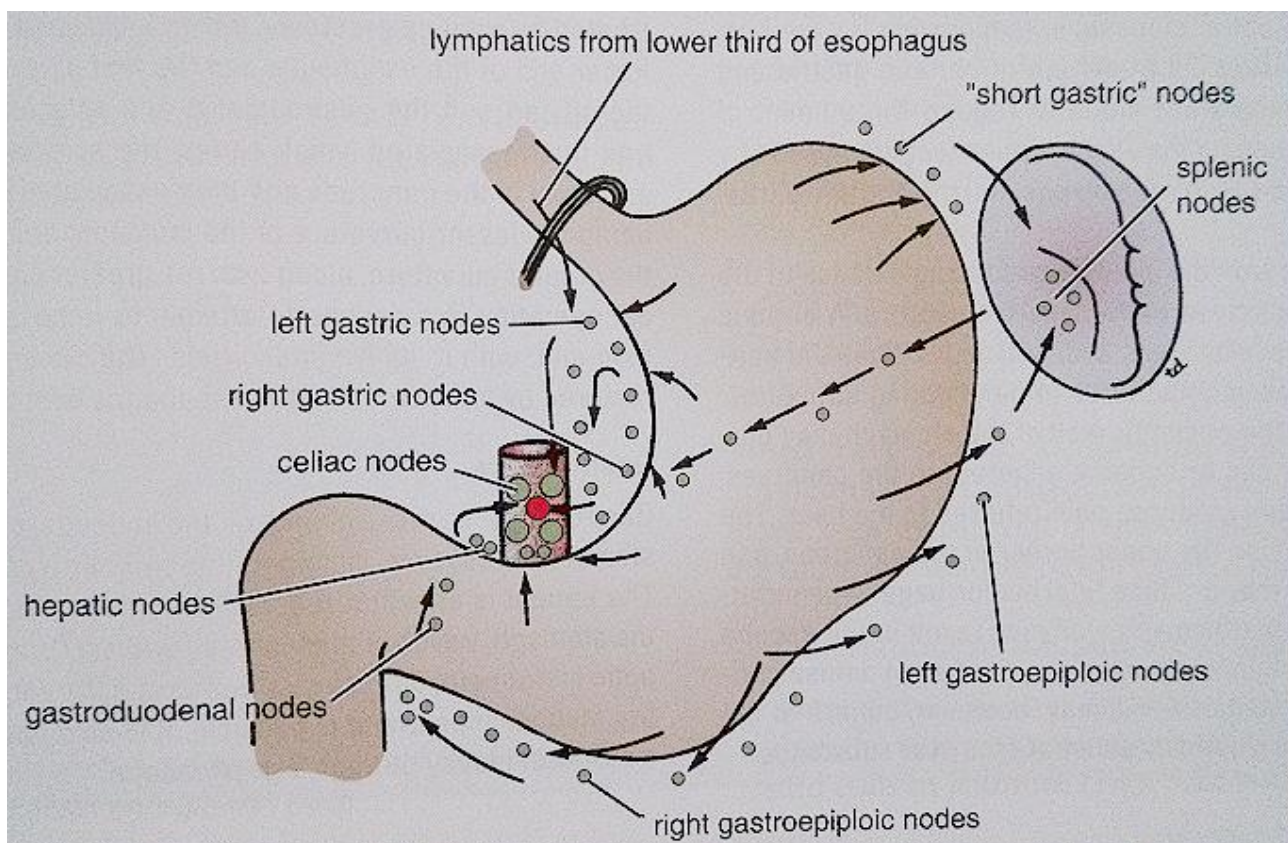
- a. The area of the stomach supplied by the right gastroepiploic artery drains into sub-pyloric nodes around the gastro duodenal continues up to the gastro – duodenal artery to the hepatic nodes along the hepatic artery and then to celiac nodes surrounding the celiac axis. This lymphatic pathway communicates with lymphatics of the pancreatico-duodenal arcades and the lymphatics at the hilum of the liver.

- b. The area of the stomach supplied by the short and left gastro – epiploic arteries drain across the gastrosplenic ligament to the pancreaticosplenic nodes in the hilum of the spleen and then through the nodes surrounding the splenic artery to the celiac nodes.

- c. The area of the stomach supplied by the left gastric artery drains to the nodes in the lesser omentum surrounding the bifurcation of the left gastric artery

and then through the nodes along the main trunk of the left gastric artery to the celiac nodes.

d. The area of the stomach supplied by the right gastric artery drains to subpyloric nodes surrounding the right gastric vessels. From these nodes, drainage continues to lymphatics surrounding the hepatic artery, which joins the lymphatics draining the right gastro-epiploic area along the hepatic artery to the celiac nodes. The celiac nodes represent the primary collecting point for all the 4 primary lymphatic pathways draining the stomach.



**FIGURE 4. THE LYMPHATIC DRAINAGE OF STOMACH.**

### **First part of the Duodenum:**

It is about 5cms long and the most movable of the 4 parts of the duodenum. It begins at the pylorus and ends at the neck of the gall bladder. It is covered with peritoneum over the whole of the anterior portion, but is devoid of peritoneum posteriorly. It is related above and in front to the quadrate lobe of the liver and gallbladder; above and posteriorly to the epiploic foramen; behind with the gastro duodenal artery, the bile duct and portal vein; and below and behind with the head and neck of the pancreas. It is supplied by the hepatic, gastro-duodenal and pancreatico duodenal arteries. The lymph vessels run anteriorly and posteriorly to end in the pyloric nodes, which are present on the anterior and posterior parts of the pancreatico-duodenal groove.

### **Microscopic features**

**Stomach:** The stomach is lined with columnar epithelium. It consists of 4 layers; serous, muscular, sub-mucous and mucous layers. The serosa or the visceral peritoneum covers the entire surface of the stomach except the bare area posteriorly and along the greater and lesser curvatures at the lines of attachment of the greater and lesser omentum, where the two layers of peritoneum leave a small space in which the vessels and nerves lie. The muscular layer is situated immediately beneath the serous layer and consists of 3 sets of muscle fibres which are the longitudinal, circular and oblique fibres. The pyloric sphincter consists of a thickening of the middle circular muscle fibres. The submucous layer consists of loose areolar tissue. The mucous

membrane is thick and its surface is smooth, soft and velvety. It has numerous folds or rugae during the contracted state which are obliterated when the stomach is distended. The luminal surface of the mucosa contains a number of depressions called the gastric pits in to which 3 to 7 gastric glands open into it.

**TABLE 1.THE GASTRIC CELL TYPES, LOCATION & FUNCTION**

<b>Cells</b>	<b>Location</b>	<b>Function</b>
Parietal	Body	Secretion of acid, ghrelin, leptin, and intrinsic factor
Mucus	Body, antrum	Mucus
Chief	Body	Pepsin and leptin
Surface epithelial	Diffuse	Mucus, bicarbonate, and prostaglandins
ECL	Body	Histamine
G	Antrum	Gastrin
D	Body, antrum	Somatostatin
Gastric mucosal interneurons	Body, antrum	Gastrin-releasing peptide
Enteric neurons	Diffuse	CGRP, others

CGRP - calcitonin gene–related peptide; ECL - enterochromaffin-like.

**Duodenum:** Long slender villi lined by columnar epithelium are characteristic. Brunner's glands are situated between the muscularis mucosa and inner muscle layer.

## **PHYSIOLOGY**

### **Gastric Secretion**

Gastric glands secrete about 250ml of gastric juice daily. The parietal cells secrete hydrochloric acid in the concentration of about 150 mmol / litre. The HCl secreted kills many ingested bacteria, aids protein digestion, provides necessary Ph for pepsin to start protein digestion and stimulates the flow of pancreatic and bile juice.

The gastro–duodenal mucosa secretes pepsinogens from the chief cells present in the oxyntic, pyloric and Brunner glands. They are inactive proenzymes which are converted into their active form below a pH of 5. The active form pepsin is a protease and acts maximally at an optimum pH of 2. The intrinsic factor is secreted by the oxyntic cells which form a complex with Vitamin B12 and in the presence of calcium ions the complex is absorbed in the terminal ileum.

The mucus secreting cells found in the body and antrum of stomach secrete mucous whose function is lubrication and providing a major protective barrier for the stomach wall against the injurious effects of the gastric juice. The Brunner glands of the duodenum secrete mucus rich in bicarbonates which help in protecting the duodenum from the effects of the gastric juice. The transport of

bicarbonate to the lumen from the surface epithelium is stimulated by prostaglandin E<sub>2</sub> which is inhibited by aspirin.

### **Gastric Mucosal Barrier:**

HCl can cause tissue damage but the following things protect the gastric mucosa

1. Mucus secretion: mucin secreted by neck and surface cells forms a flexible gel layer which exhibits a diffusion coefficient for H<sup>+</sup> that is one-fourth that of H<sub>2</sub>O.
2. Bicarbonate secretion: epithelial cells secrete HCO<sub>3</sub><sup>-</sup> creating an essentially neutral environment immediately adjacent to cell surface.
3. Epithelial barrier: Tight junctions prevent backflow of HCl.
4. Mucosal blood flow: Rich blood supply removes back diffused acid and provides O<sub>2</sub>, HCO<sub>3</sub><sup>-</sup> and nutrients.

Substances that are soluble in lipids and non-ionized at acidic pH readily penetrate this barrier causing its break-down and destroy the mucous cells. They include aspirin, ethanol above 10%, detergents bile salts, lysolecithin etc. When the barrier is broken down, there is back diffusion of acid through the gastric mucosa which results in increased gastric motility, increased acid and pepsinogen production.

Histamine is released from the mast cells present in the mucosa. The mucosal blood flow and capillary permeability are increased resulting in mucosal edema. The damage to the surface mucosal cells and increased capillary permeability

cause a leakage of protein and plasma into the lumen. The mucosal blood vessel may be destroyed producing haemorrhage.

### **Gastric Motility and Emptying:**

Gastric pace maker is present in circular fibres of the fundus. When the stomach is filled, weak constrictor waves or mixing waves move towards the antrum along the stomach wall. As these waves progress towards the antrum they increase in intensity and force the antral contents towards the pylorus. During each peristalsis

only a fraction of the antral contents are expelled into the duodenum while the rest is squirted back resulting in further mixing of the antral contents. Stomach emptying is promoted by peristaltic waves in the antrum and is opposed by resistance of the pylorus to the passage of food. The rate at which the stomach empties is regulated by signals from the stomach and duodenum. The stomach signals are (a) nervous signals caused by distension of food and (b) release of gastrin from the stomach. Both these signals increase the pyloric pumping force and at the same time inhibit the pylorus, thus promoting gastric emptying. The signals from the duodenum initiate the entero-gastric reflex which results in depression of the pyloric pump and increases the tone of the pylorus.

### **Phases of Gastric Secretion:**

- A. Cephalic Phase: stimulation of anterior hypothalamus and parts of adjacent orbital frontal cortex increases vagal efferent activity by sight, smell or thought. Accounts for one-third to half of gastric secretion.

B. Gastric Phase: In response to stretch and chemical stimuli (amino acids and other products of digestion) to receptors in the wall of stomach there will be acid and gastrin secretion through reflex arc situated totally in stomach.

C. Intestinal Phase: Presence of food in the duodenum causes the stomach to secrete small amounts of gastric juice secondary to gastrin secretion from the duodenum.

### **Regulation of Gastric Secretion:**

The gastric secretion is stimulated by vagal activation, gastrin and gastric histamine. Vagus is activated by (a) Cephalic stimuli (b) distension of stomach and (c) hypoglycaemia. Gastrin is released secondary to (a) Vagal activation (b) distension of stomach (intra-mural reflex) (c) substances like ethanol, amino acids, digested proteins etc. (chemical stimuli) and (d) humoral agents like epinephrine and bombesin.

The gastric secretion is inhibited by (a) blocking the release of gastrin which is brought about by acidification of antrum and interaction by other hormones like secretin, glucagons and calcitonin ; (b) release of gastric inhibitory peptide by the gastric mucosa and (c) entero gastric reflex which is initiated by the intramural plexus, sympathetic nerves and the vagi secondarily to presence of acid and protein break-down products in the upper intestine, distension of the small bowel and irritation of mucosa.



## AETIOLOGY

Gastric outlet obstruction is caused by various pathological conditions caused by varied aetiologies

### A. Congenital

- Congenital hypertrophic pyloric stenosis
- Pyloric atresia/pyloric mucosal diaphragm
- Duodenal atresia/stenosis
- Malrotation/incomplete rotation
- Annular pancreas/heterotopic pancreatic tissue
- Duplication of stomach and/or duodenum.

### B. Acquired

#### I. Inflammatory

- Chronic cicatrizing duodenal ulcer
- Gastroduodenal tuberculosis
- Strictures due to corrosives, curling's ulcer or post-operative scarring
- Pancreatic pseudocyst
- Gastric syphilis
- Crohn's disease
- Adhesions due to chronic cholecystitis or post-cholecystectomy
- Post-radiation gastritis
- Cholecystitis

- Acute pancreatitis

## II. Neoplastic

- Carcinoma stomach (pylorus)
- Benign tumours of stomach and duodenum
  - Epithelial polyp
  - Mesenchymal neoplasm
  - Miscellaneous tumours
- Malignant mesenchymal tumours
- Other malignancies
  - Carcinoma of pancreas
  - Carcinoid tumours
  - Sarcoma
  - Melanoma

## III. Miscellaneous

- Hypertrophic pyloric stenosis in adults
- Wilkie's syndrome (duodenal ileus)
- Foreign bodies and bezoars
- Intramural hematoma
- Gall stone obstruction of duodenum
- Crohn's disease
- Aberrant vessels

- Duodenal diverticula
- Paraduodenal hernia
- Radiation induced, especially second part of duodenum
- Chemotherapy induced – continuous hepatic artery infusion of 5 FU

## **AETIOPATHOGENESIS**

### **I. INFLAMMATORY CAUSES OF GASTRIC OUTLET OBSTRUCTION**

#### **A. Gastric Outlet Obstruction Secondary to Chronic Duodenal Ulcer:**

Gastric outlet obstruction occurs in no more than 5% of patients with peptic ulcer disease. It is usually due to duodenal or prepyloric ulcer disease, and may be acute (from inflammatory swelling and peristaltic dysfunction) or chronic (from cicatrix). The pathogenesis of duodenal ulcer is not clear and no single theory explains all types of lesions.

The various factors involved in ulcer formation include

- a. Hyper - secretion of acid which is associated with 40% of the cases;
- b. Impaired mucosal defence caused by various agents like NSAID, smoking, stress and H. Pylori even though the acid secretion is within normal limits.
- c. Genetic factor – there is evidence to show that the duodenal ulcer run in families and 40% of the people with this autosomal dominant characteristic develop duodenal ulcer.

Because of ulceration there may be pylorospasm, edema, inflammation or dysmotility which are reversible. Irreversible changes like fibrosis, scarring and deformity also underlie. The obstruction is caused by chronic cicatrization of the duodenal ulcer in which the scar contracture gradually narrows the lumen. There is greater destruction of muscular coat than mucosa. Surrounding arteries may show the features of endarteritis obliterans.

Four histological zones are described surrounding chronic peptic ulcer – superficial layer of fibrin and exudates with successive underlying zones of fibrinoid necrosis, granulation tissue and fibrosis.

### **HELICOBACTER PYLORI**

With specialized flagella and a rich supply of urease, *H.pylori* is uniquely equipped for survival in the hostile environment of the stomach. The sequence of inflammation to metaplasia to dysplasia to carcinoma, is now increasingly well recognized to occur in the stomach with *Helicobacter*-induced gastritis. The organism possesses the enzyme urease, which converts urea into ammonia and bicarbonate, thus creating an environment around the bacteria that buffers the acid secreted by the stomach. The ammonia is damaging to the surface epithelial cells. The organism lives in the mucus layer atop the gastric surface epithelial cells, and some attach to these cells.

The other mechanisms by which *Helicobacter* causes gastric injury may be

- inhibitory effect on antral D cells that secrete somatostatin, a potent inhibitor of antral G cell gastrin production
- production of toxins (vacA and cagA),
- local elaboration of cytokines (particularly interleukin-8) by infected mucosa,
- recruitment of inflammatory cells and release of inflammatory mediators,
- recruitment and activation of local immune factors, and increased apoptosis

Up to 90% of patients with duodenal ulcers, and 70 to 90% of patients with gastric ulcers, have *H. pylori* infection

## **B. Gastroduodenal Tuberculosis**

Involvement of the stomach and duodenum is rare; an autopsy series has reported an incidence around 0.5%. With HIV infection, the incidence is on rise even in developed countries. Possible causes for GD sparing include high acidity, a paucity of lymphoid tissue and rapid transit of food in the stomach. Usually the ulcer occurs at the junction of first and second part of duodenum. Gastric involvement most likely originates from adjacent celiac lymph nodes. Obstruction is the most common cause of presentation, and occurs in the hypertrophic form or involves perigastric or periduodenal lymph nodes with subsequent fibrosis.

### **C. Corrosive Strictures**

In most of the cases of corrosive poisoning, oesophagus escapes from the effect of corrosives and gastric outlet obstruction develops in about 1-6 weeks after ingestion of corrosives. Hydrochloric acid, nitric acid, sulphuric acid, carbolic acid, ferrous sulphate, copper sulphate, formaldehyde, tincture iodine, etc are the corrosives causing obstruction.

### **D. Post – operative adhesions**

Adhesions of the stomach are less common than that of other parts. The common surgery causing post – operative adhesion leading to gastric outlet obstruction is cholecystectomy because of the formation of band between pyloroduodenal junction and liver surface resulting in kinking. The obstruction may be partial or complete. Even cholecystitis can cause adhesion between pylorus, omentum and liver bed. Gastric outlet obstruction with history of fatty dyspepsia should be investigated to rule out cholecystitis.

### **E. Crohn's disease**

A rare cause of gastric outlet obstruction usually associated with ileal disease.

### **F. Pseudo pancreatic cyst**

Most of the times it is found in lesser sac, behind the stomach. When large enough, causes obstruction to gastric outlet by mechanical pressure.

### **G. Post – radiation gastritis**

Radiation tolerance of stomach is high and gastritis occurs occasionally. Usually 4000 rads are delivered over a period of 3 – 4 weeks. If it exceeds, then radiation gastritis occurs and may lead to gastric ulceration, fistula or antral stenosis.

## **II. NEOPLASTIC CAUSES OF GASTRIC OUTLET OBSTRUCTION**

### **A. Carcinoma Stomach**

Adenocarcinoma of the stomach was the leading cause of cancer-related death worldwide through most of the 20th century. It now ranks second only to lung cancer. The aetiological factors can be divided into

#### **1. Acquired factors**

##### **a. Nutritional**

High salt consumption

High nitrate consumption

Low dietary vitamin A and C

Poor food preparation (smoked, salt cured)

Lack of refrigeration

Poor drinking water (well water)

##### **b. Occupational**

Rubber workers

Coal workers

- c. Cigarette smoking**
- d. Helicobacter pylori infection**
- e. Epstein-Barr virus**
- f. Radiation exposure**
- g. Prior gastric surgery for benign gastric ulcer disease**

## **2. Genetic factors**

Type A blood

Pernicious anaemia

Family history

Hereditary non polyposis colon cancer

Li-Fraumeni syndrome

## **3. Precursor lesions**

Adenomatous gastric polyps

Chronic atrophic gastritis

Dysplasia

Intestinal metaplasia

Menetrier's disease

Gastric adenocarcinomas of the body and antrum of the stomach have a strong association with *H. pylori* infection. This is a common infection in many parts of the world and was associated with a doubled risk of such cancers in a meta-analysis of multiple studies. The precise mechanism by which *H. pylori* infection increases gastric cancer incidence is unclear, but it appears to increase



the incidence of chronic atrophic gastritis, which produces a low-acidity environment, and the incidence of metaplasia and dysplasia.

There is fairly strong evidence that eating fruits and vegetables (especially raw) has a protective effect against gastric cancer, and there is a suggestion that eating foods high in antioxidants including vitamins C and E, carotenoids, and flavonoids may be beneficial.

## **Pathological Classification**

### **A. Lauren's classification**

In 1965, Lauren described two histologic types of gastric adenocarcinoma

1. The intestinal variety represents a differentiated cancer with a tendency to form glands. The intestinal variant arises from precancerous lesions such as gastric atrophy or intestinal metaplasia within the stomach; occurs more commonly in men than in women; is more frequent in older people; and represents the dominant histologic type in regions where stomach cancer is endemic, suggesting a predominantly environmental aetiology.
2. The diffuse form exhibits very little cell cohesion and has a predilection for extensive submucosal spread and early metastases. The diffuse form does not typically arise from recognizable precancerous lesions. It is more common in low-incidence regions, occurs slightly more frequently in women and in younger patients, and has a higher association with familial occurrence (blood group A), suggesting a genetic aetiology.

## **B. Borrmann's classification**

The Borrmann classification divides gastric cancer into five types depending on macroscopic appearance.

Type I - polypoid or fungating cancers

Type II - ulcerating lesions surrounded by elevated borders,

Type III - ulcerated lesions infiltrating the gastric wall,

Type IV - diffusely infiltrating tumours

Type V - unclassifiable cancers.

## **C. Broder's classification:** This is based on histology

1. Well differentiated
2. Moderately differentiated
3. Poorly differentiated
4. Anaplastic

## **D. Ming's classification:** Histomorphologic staging system

1. Expansive type – with a favourable prognosis. The expansive-type tumours were uniformly polypoid or superficial on gross appearance.
2. Infiltrating type – with a poor prognosis. The infiltrative tumours were almost always diffuse on gross appearance.

About 95% of gastric carcinomas are adenocarcinoma, which are further divided into

- Papillary
- Tubular

- Mucinous
- Signet ring

Mucinous and signet ring cell tumours have poor prognosis

The Japanese Research Society for Gastric Cancer has classified early gastric cancers (EGC) based on endoscopic criteria first established by the Japanese Endoscopy Society for the description of T1 tumours. The current classification system is used for both in situ and invasive tumours and categorizes tumours based on endoscopic findings as follows:

Type I:- protruded

type IIa:- superficial elevated

type IIb:- flat

type IIc:- superficial depressed

type III:- excavated,

### **Modes of spread**

The initial growth of the tumour occurs by penetration into the gastric wall, extension through the wall, and involvement of an increasing percentage of the stomach. The two modes of local extension that can have a major therapeutic impact are tumour penetration through the gastric serosa, where the risk of tumour invasion of adjacent structures or peritoneal spread is increased, and involvement of lymphatics. Tumour spread is often through the intramural lymphatics or in the subserosal layers. The modes of spread are

1. Direct: Local extension does not occur solely by radial intramural spread but also by deep invasion through the wall to involve adjacent structures. Extension can occur through the gastric serosa to involve omentum, spleen, adrenal gland, diaphragm, liver, pancreas, or colon. Local extension can also occur into the oesophagus or the duodenum. Duodenal extension is principally through the muscular layer by direct infiltration and through the subserosal lymphatics, but is not generally of great extent. Extension into the oesophagus occurs primarily through the submucosal lymphatics.
2. Lymphatic spread: spread to draining lymph nodes in adjacent areas. Spread along ligamentum teres produces hard, knobby, red tumour (Sister Mary Joseph's nodule). Left supraclavicular node involvement occurs via thoracic duct in late cases (Troisier's sign)
3. Transcoelomic spread: Once the serosa is breached the cells are freely discharged into peritoneal cavity causing peritoneal malignancy and ascites. These tumour deposits may be felt through rectal examination (Blummer's shelf). The cells may get implanted on raw surface created over an active ovary in premenopausal women causing Krukenberg's tumour.
4. Haematogenous spread: The spread may occur to liver, lungs, peritoneum, omentum, mesentery, pancreas, adrenal glands, bones and skin.

## **Other malignancies of stomach**

### **I. Gastric carcinoids**

These account for less than 2% of all carcinoid tumours as compared with 70% in appendix and 20% in ileum and caecum.

### **II. Malignant smooth muscle tumours**

These comprise of 1-2% of all malignant lesions of stomach. The common conditions are leiomyoblastoma and leiomyosarcoma. Other mesenchymal tumours are neurofibrosarcoma, Kaposi's sarcoma, fibrosarcoma, haemangiopericytoma and rhabdomyosarcoma.

### **B. Duodenal malignancies**

Carcinoma of first part of duodenum is rare. The incidence is maximum at periampullary region. Both ulcerative and papilliferous growth can cause duodenal obstruction. Other tumours of duodenum are carcinoids, melanomas and sarcomas, which are still rarer.

### **C. Carcinoma head of the pancreas**

Accounts for about 5% of cancer related deaths. Because of difficulties in diagnosis, the aggressiveness of pancreatic cancers and the lack of effective systemic therapies, generally fewer than 5% of patients with adenocarcinoma of the pancreas survive 5 years after diagnosis. Thus incidence rates and mortality rates are identical. About 5-8% of pancreatic cancers have familial predisposition. These include MEN-I syndrome, hereditary pancreatitis, Lynch syndrome of hereditary nonpolyposis colon cancer, ataxia telangiectasia and the

familial atypical multiple mole melanoma syndrome. Chronic pancreatitis, tobacco smoking and diet rich in fat and protein are important etiological factors.

#### **D. Benign tumours of the stomach**

These account for less than 2% of all gastric neoplasms

##### **1. Epithelial polyps**

- i. Adenomatous polyps – common in antrum
- ii. Hyperplastic polyps – these are distributed throughout the stomach and constitute for 75% of all gastric epithelial polyps.

##### **2. Mesenchymal neoplasms**

These are common in distal stomach and obstruction may be produced by prolapse of the tumour into duodenum. Though fibromas, schwannomas, neurofibromas, lipomas, etc occur, leiomyomas are the commonest.

#### **E. Gastric lymphomas**

Primary gastric lymphoma is the commonest site of extranodal NHL and constitutes 2% of all gastric malignancies. They are common in posterior wall and along lesser curvature.

### **III. Miscellaneous causes of gastric outlet obstruction**

#### **a. Adult pyloric stenosis**

A rare condition, whose relationship to childhood condition is unclear, although some patients have a long history of problems with gastric emptying. Normal pyloric canal measures 0.5cm to 1cm in length and thickness of its musculature ranging from 3 to 9 mm with an average of 5.5mm. The longitudinal muscles are not affected by the disease. There may be moderate thickening due to effects of obstruction

#### **b. Pyloric mucosal diaphragm**

The origin of this rare condition is unknown. It usually does not become apparent until middle life.

#### **c. Duodenal ileus**

It is also called as Wilkie's syndrome, gastromesenteric ileus, arteriomesenteric ileus, superior mesenteric artery syndrome. The third part of the duodenum, as it crosses the spine is fixed in compartment rounded posteriorly by spine and aorta and anteriorly by root of mesentery containing superior mesenteric nerves and vessels. The causes are increased lordosis, a short mesentery, narrow aorto-mesenteric root, short length and high attachment of ligament of Treitz, marked loss of weight may decrease aorto-mesenteric angles.

#### **d. Bezoars**

Phytobezoars may occur in patients after gastric surgery (because of reduced peristalsis, hypochlorhydria, inadequate chewing, high fibre intake, loss of gastric pump mechanism). Trichobezoars (hair balls) are unusual and are virtually exclusively found in female psychiatric patients, often young.

#### **e. Foreign bodies**

Usually long foreign bodies get stuck at gastric outlet as they cannot get through fixed curvature. These could have been swallowed purposefully or accidentally.

#### **f. Bouveret's syndrome**

This is the impaction of gall stones at duodenal bulb. It is more common in elderly women.

### **CLINICAL FEATURES**

Clinical features common to all conditions causing gastric outlet obstruction can be considered as general and the symptoms pertaining to specific disease can be considered later.

#### **I. General clinical features**

##### **Symptoms**

##### **➤ Pain**

Pain or discomfort in upper abdomen especially after taking food is present in most of the times. It is dull aching and relieved by vomiting.



➤ **Vomiting**

It is a constant feature and in its absence the diagnosis of gastric outlet obstruction is to be reconsidered. Typically the vomiting is projectile and effortless, large in amount and may contain food particles ingested 2 – 3 days previously. It may be foul smelling because of fermented food particles because of increased infection which is due to hypoacidity which can be a result of chronic gastritis.

➤ **Loss of appetite**

It is seen in late cases with advanced stenosis, and profound in cases of malignancies.

➤ **Loss of weight**

Improper digestion, vomiting and loss of appetite lead to loss of weight.

➤ **Haemetamesis & melena**

It is due to longstanding gastric ulcers, chronic gastritis, smoking, alcohol consumption drugs like NSAIDs, increased intake of caffeine, emotional stress and gastric tumours. |

➤ **Constipation**

Because of repeated vomiting and dehydration, recent onset of constipation is a common complaint.

➤ **Ball rolling movement**

Patients may sometimes attribute gastric peristalsis to some lump moving in the abdomen which occurs usually after taking food.

**On examination**

➤ **Anaemia**

Due to improper nourishment (deficiency of vitamins and minerals) and due to haematemesis and melena

➤ **Dehydration**

Dehydration commences if the fluid losses are over 6% of body weight and here it is because of vomiting there will be loss of water,  $K^+$ ,  $Na^+$ ,  $Cl^-$ .

➤ **Visible gastric peristalsis**

It is a ball-rolling movement in upper abdomen because of gastric peristalsis from left to right. Typically it starts in the left hypochondrium, crosses the midline and ends in the right hypochondrium. Clinically it is elicited after making the patient drink as much water he can.

➤ **Dilated stomach**

In most of the cases of gastric outlet obstruction stomach will be dilated below the level of umbilicus. In advanced cases it can be in the pelvis. Following methods can be adopted to make out the dilatation of stomach.

➤ **Percussion method**

The area of stomach is percussed centrifugally and greater curvature is marked at the point of change of note.

➤ **Auscultoscraping**

Bell of stethoscope is placed just below and left of xiphisternum. Abdomen is scraped radially and centrifugally with a blunt object and the points of change of note mark the greater curvature.

➤ **Succussion splash**

Performed three hours after food, by hearing for the gurgle produced by shaking the patient after keeping the ear close to anterior abdominal wall.

## **II. SPECIFIC CLINICAL FEATURES**

### **A. Cicatrised Peptic Ulcer**

➤ **Vomiting**

The patient usually will have non – bilious painless vomiting once in the evening. In advanced cases vomiting may occur at any time.

➤ **Pain**

The pain is epigastric, often described as gnawing, and may radiate to the back. Eating may sometimes relieve the discomfort. The pain is normally intermittent rather than intractable.

➤ **Periodicity**

One of the classic features of untreated peptic ulceration is periodicity. Symptoms may disappear for weeks or months to return again. This periodicity may be related to the spontaneous healing of the ulcer

➤ **Features of nutritional disturbances**

Vomiting and dehydration may cause anorexia, coated tongue, thirst, unpleasant taste in the mouth and weakness. On examination, the patient may have typical “ulcer facies” and mentally confused and have tetany because of electrolyte imbalance. Skin is dry, wrinkled and turgor is lost.

A history of previous GI bleed may be obtained.

**B. Gastroduodenal tuberculosis**

Usually this is associated with pulmonary or intestinal tuberculosis whose clinical features may be elicited.

**C. Gastric outlet obstruction due to corrosives**

There will be history of consumption of corrosives

**D. Gastric outlet obstruction due to post – operative adhesions**

There will be history of surgery and one will find operative scar over the abdomen. Patient may give history of acute abdominal pain suggesting obstruction.

### **E. Gastric outlet obstruction due to pancreatic pseudocyst**

Patient may give history of abdominal trauma or acute abdomen. Patient may be an alcoholic. On examination, a mass may be felt in epigastric region, may move with respiration, best seen when viewed from sides. It is round, smooth, usually tense and resonant as it is covered by stomach and nasogastric tube may be felt superficial to the stomach.

### **F. Crohn's disease**

General features of gastric outlet obstruction may be accompanied with intermittent pyrexia, diarrhoea and steatorrhoea.

### **G. Carcinoma stomach**

Because of the vague, nonspecific symptoms that characterize gastric cancer, most patients are diagnosed with advanced-stage disease. Patients may have a combination of signs and symptoms such as weight loss, anorexia, fatigue, or epigastric discomfort, none of which unequivocally indicates gastric cancer.

Weight loss is a common symptom. Early satiety is an infrequent symptom of gastric cancer but is indicative of a diffusely infiltrative tumour that has resulted in loss of distensibility of the gastric wall. Persistent vomiting is consistent with an antral carcinoma obstructing the pylorus. Significant gastrointestinal bleeding is uncommon with gastric cancer; however, hematemesis does occur in approximately 10% to 15% of patients. Ascites, jaundice, or a palpable mass indicates extensive and incurable disease. Signs

and symptoms at presentation are often related to spread of disease. Because the transverse colon is held in proximity to the stomach by the gastrocolic ligament, the transverse colon is a potential site of malignant fistulisation and obstruction from a gastric primary tumour. Diffuse peritoneal spread of disease frequently produces other sites of intestinal obstruction. A large ovarian mass (Krukenberg's tumour) or a large peritoneal implant in the pelvis (Blummer's shelf), which can produce symptoms of rectal obstruction, may be felt on pelvic or rectal examination. Nodular metastases in the subcutaneous tissue around the umbilicus (Sister Mary Joseph's nodules) or in peripheral lymph nodes represent areas in which a tissue diagnosis can be established with minimal morbidity. Left supraclavicular (Virchow's) or left axillary (Irish) lymphnodes may be palpable or migratory thrombophlebitis (Trousseau's sign) may be present.

### **Staging**

Currently, the most widely use staging system is the AJCC, TNM staging system. This is based on the depth of tumour invasion (T), number of involved lymphnodes (N), and presence or absence of metastatic disease (M). In the current staging system, a minimum of 15 nodes must be evaluated for accurate staging. Some have suggested that other factors be included in the T and N assessment, such as the location of the primary (cardia compared with distal tumours), because this may independently predict survival, and emphasis on the percentage of positive nodes (lymph node ratio) rather than the number of

positive nodes. However, the current AJCC staging system does not reflect these factors.

Although not part of the formal AJCC staging system, the term *R status*, first described by Hermanek in 1994, is used to describe tumour status after resection and is important for determining the adequacy of surgery. R0 describes a microscopically margin-negative resection, in which no gross or microscopic tumour remains in the tumour bed. R1 indicates removal of all macroscopic disease, but microscopic margins are positive for tumour. R2 indicates gross residual disease. Because the extent of resection can influence survival, some include this R designation to complement the TNM system. Long-term survival can be expected only after an R0 resection.

The AJCC system is not specific for nodal location. In the previous version of the Union Internationale Contre le Cancer (UICC) TNM system, N categories were defined by the location of lymph node metastases relative to the primary, with pN1 defined as positive nodes 3 cm or less from the primary and pN2 as more than 3 cm from the primary or nodal metastases along named blood vessels.

## **TNM Classification of Carcinoma of the Stomach**

### **PRIMARY TUMOR (T)**

TX - Primary tumour cannot be assessed

T0 - No evidence of primary tumour

Tis - Carcinoma in situ; intraepithelial tumour without invasion of the lamina propria

T1 - Tumor invades lamina propria, muscularis mucosa, or submucosa

T1a - Tumor invades lamina propria or muscularis mucosa

T1b - Tumor invades submucosa

T2 - Tumor invades muscularis propria

T3 - Tumor penetrates subserosal connective tissue without invasion of visceral peritoneum or adjacent structures

T4 - Tumor invades serosa (visceral peritoneum) or adjacent structures

T4a - Tumor invades serosa (visceral peritoneum)

T4b - Tumor invades adjacent structures

## **REGIONAL LYMPH NODES (N)**

NX - Regional lymph node(s) cannot be assessed

N0 - No regional lymph node metastasis

N1 - Metastasis in 1-2 regional lymph nodes

N2 - Metastasis in 3-6 regional lymph nodes

N3 - Metastasis in 7 or more regional lymph nodes

N3a - Metastasis in 7-15 regional lymph nodes

N3b - Metastasis in 16 or more regional lymph nodes

## **DISTANT METASTASIS (M)**

M0 - No distant metastasis

M1 - Distant metastasis



**TABLE 2. ANATOMIC STAGING OF CARCINOMA STOMACH**

0	Tis	N0	M0
IA	T1	N0	M0
IB	T2	N0	M0
	T1	N1	M0
IIA	T3	N0	M0
	T2	N1	M0
	T1	N2	M0
IIB	T4a	N0	M0
	T3	N1	M0
	T2	N2	M0
	T1	N3	M0
IIIA	T4a	N1	M0
	T3	N2	M0
	T2	N3	M0
IIIB	T4b	N0	M0
	T4b	N1	M0

	T4a	N2	M0
	T3	N3	M0
IIIC	T4b	N2	M0
	T4b	N3	M0
	T4a	N3	M0
IV	Any T	Any N	M1

## **JAPANESE GASTRIC CANCER ASSOCIATION STAGING SYSTEM FOR GASTRIC CANCER**

### **TUMOR STAGE**

T1 Tumor invasion of mucosa and/or muscularis mucosa (M) or submucosa (SM)

T2 Tumor invasion of muscularis propria (MP) or subserosa (SS)

T3 Tumor penetration of serosal (SE)

T4 Tumor invasion of adjacent structures (SI)

TX Unknown

### **NODAL STAGE**

N0 No evidence of lymph node metastasis

N1 Metastasis to group 1 lymph nodes, but no metastasis to group 2 to 3 lymphnodes

N2 Metastasis to group 2 lymph nodes, but no metastasis to group 3 lymph nodes

N3 Metastasis to group 3 lymph nodes

NX Unknown

### **HEPATIC METASTASIS STAGE (H)**

H0 No liver metastasis

H1 Liver metastasis

HX Unknown

### **PERITONEAL METASTASIS STAGE (P)**

P0 No peritoneal metastasis

P1 Peritoneal metastasis

PX Unknown

### **PERITONEAL CYTOLOGY STAGE (CY)**

CY0 Benign/indeterminate cells on peritoneal cytology

CY1 Cancer cells on peritoneal cytology

CYX Peritoneal cytology was not performed

### **OTHER DISTANT METASTASIS (M)**

M0 No other distant metastases (although peritoneal, liver, or cytological metastases may be present)

M1 Distant metastases other than the peritoneal, liver or cytological metastases

MX Unknown

The Japanese Classification for Gastric Carcinoma (JCGC) staging system describes the anatomic locations of nodes removed during gastrectomy. Sixteen distinct anatomic locations of lymph nodes are described, with the recommendation for nodal basin dissection dependent on the location of the primary. The lymph node stations, or echelons, are numbered and further classified into groups of echelons corresponding to the location of the primary and reflect the likelihood of harbouring metastases. The presence of metastasis to each lymphnode group then determines the N classification. For example, metastasis to any of the group 1 lymph nodes in the absence of disease in more distant lymph node groups is classified as N1.

**TABLE 3. LYMPH NODE STATION NUMBERS (GROUPS 1-3) BY LOCATION OF PRIMARY TUMOUR**

		<b>LOCATION OF PRIMARY TUMOUR</b>		
<b>Lymph node station (No.)</b>	<b>Description</b>	<b>Upper third</b>	<b>Middle third</b>	<b>Lower Third</b>
1	Right paracardial	1	1	1
2	Left paracardial	1	3	M
3	Lesser curvature	1	1	1
4sa	Short gastric	1	3	M
4sb	Left gastroepiploic	1	1	3

4d	Right gastroepiploic	2	1	1
5	Suprapyloric	3	1	1
6	Infrapyloric	3	1	1
7	Left gastric artery	2	2	2
8a	Anterior common Hepatic	2	2	2
8p	Posterior common hepatic	3	3	3
9	Celiac artery	2	2	2
10	Splenic hilum	2	3	M
11p	Proximal splenic	2	2	2
11d	Distal splenic	2	3	M
12a	Left hepatoduodenal	3	2	2
12b,p	Posterior hepatoduodenal	3	3	3
13	Retropancreatic	M	3	3
14v	Superior mesenteric Vein	M	3	2
14a	Superior mesenteric Artery	M	M	M
15	Middle colic	M	M	M
16a1	Aortic hiatus	3	M	M
16a2,16b1	Para-aortic, middle	M	3	3
16b2	Para-aortic, caudal	M	M	M

M - Lymph nodes regarded as distant metastasis.

## **F. Benign tumours of stomach**

Atrophic gastritis is usually associated with hyperplastic polyps. Epithelial polyps may present with epigastric pain, haematemesis or melena. Gastric leiomyoma presents with haematemesis or melena. Bleeding from tumour may be massive or intermittent.

## **G. Gastric lymphoma**

Abdominal pain occurs in more than 80% of patients. It may be associated with early satiety, nausea, night sweats, fever & haemorrhage. On examination, a mass may be felt in left upper abdomen. Splenomegaly may be present.

## **H. Duodenal malignancy**

Though they are rare may present with obstruction as opposed to periampullary carcinoma which slough off before obstruction is developed. On examination there may be jaundice and palpable mass in right upper quadrant.

## **I. Carcinoma head of the pancreas**

Two-thirds of the cases present with painless, progressive jaundice. Usually associated with pruritis, pale coloured stools and dark coloured urine. Epigastric pain may radiate to back due to involvement of celiac and mesenteric plexus. In 30% of cases gall bladder may be palpable and liver in 50% of cases. In cases of splenic vein thrombosis, spleen may be enlarged. Exocrine insufficiency due to duct obstruction may result in malabsorption and steatorrhoea. Glucose intolerance is present in the majority of patients with

pancreatic cancer, because of altered  $\beta$  – cell function and impaired tissue insulin sensitivity. Symptoms of gastric outlet obstruction are due to external compression or by direct invasion of pyloric antrum or second part of duodenum.

### **J. Duodenal ileus**

Though no age is exempt, it usually occurs in young adults. Acute presentation is less common and may be precipitated by application of plaster cast or bed rest in supine position. Chronic duodenal ileus presents with epigastric pain, fullness after meals and foul eructation. Symptoms may get relieved on knee – chest position or left lateral position. Patient may be thin and asthenic. Epigastric pulsations may be present.

### **K. Foreign bodies**

Large foreign bodies, which fail to negotiate through gastric outlet may present with features of obstruction due to ball – valve mechanism, while long foreign bodies obstruct the outlet mechanically and there may be ulceration and pylorospasm. It may present with hematemesis and malena.

## **Complications of gastric outlet obstruction**

### **1. Metabolic effects**

As a result of the loss of gastric juice in vomitus or even its sequestration within the dilated stomach profound metabolic disturbances may occur. Gastric juice contains an average of 100 meq/L of  $\text{Cl}^-$ , 45 meq/L of  $\text{Na}^+$ , and 10 meq/L of  $\text{K}^+$ . Initially vomiting causes loss of hydrochloric acid in excess of sodium

and potassium. The hydrogen ions are derived from carbonic acid with residual bicarbonate passing into ECF. So, there will be fall in plasma  $\text{Cl}^-$  and rise in  $\text{HCO}_3^-$ . In early stages, this alkalotic tendency is compensated for by renal limits. It is of interest that there is little evidence of respiratory compensation or excretion of this excess of carbon dioxide in expired air. Urine is alkaline because of diminished chloride content and increased bicarbonate and is concentrated because of reduced volume because of dehydration.

In long standing cases, because of loss of large amounts of gastric juice that is rich in hydrogen chloride and  $\text{K}^+$ , there is hypokalemic, hypochlorhydric alkalosis. Sodium deficit increases as loss is both in vomitus and in urine. In urine it is excreted with bicarbonate which must be accompanied by a cation. This causes decrease in ECF which sometimes can cause shock. This sodium deficit stimulates aldosterone secretion which conserves sodium at the cost of  $\text{K}^+$  and  $\text{H}^+$ . There will be Pre – renal azotemia as a result of decreased GFR and urea clearance.  $\text{H}^+$  excretion in urine in place of  $\text{Na}^+$  in an alkalotic condition is called as “paradoxical aciduria”. As potassium is lost from the cells, it is replaced by sodium and hydrogen ions which produce an intra – cellular acidosis and aggravates further the extracellular alkalosis.

Further, “gastric tetany” can result from a shift of weakly alkaline ionized calcium phosphate to its unionized form in an attempt to reduce alkalosis. So, there will be apparent fall in plasma calcium ion levels although total calcium



levels are normal. Because of dehydration, the concentration of electrolytes tends to increase and  $K^+$  and  $Cl^-$  levels tend to decrease in some instances, the degree of metabolic alkalosis is better reflected by level of standard bicarbonate level than electrolyte analysis.

Isotonic saline replacement is primarily needed for the correction of dyselectrolytemia. It restores the volume of ECF and plasma, and hence renal blood flow. It helps the kidney to excrete alkaline urine. Potassium can be supplemented in severe cases and dilute HCL in very severe cases.  $K^+$  supplementation of  $>20\text{meq/hour}$  requires strict cardiac beat monitoring.

Clinical presentation:-

Along with features of dehydration, there will be drowsiness, muscular hypotonia, loss of reflexes, urinary incontinence, cheyne - respiration, fall in BP with bounding pulse, sometimes tetany and abdominal distension due to paralytic ileus may be present.

## **2. Gastritis**

It is due to stasis and fermentation of gastric contents.

## **3. Gastric ulcer.**

Due to stasis and gastritis, lesser curve ulcers are seen. There may be dependent on intracellular shift of  $K^+$  and  $Mg^{2+}$ . Effortless or profuse vomiting in late stages of obstruction may be well caused by this failure in gastric peristalsis.

## **INVESTIGATIONS**

### **Blood examination**

#### **a. Hb%**

Anaemia is present in majority of the patients and is due to undernourishment, hematemesis and melena. It is microcytic, hypochromic due to loss of blood and iron deficiency but in carcinoma stomach where it is megaloblastic where it is due to intrinsic factor deficiency.

#### **b. Blood Grouping**

Persons with blood group O are about 3 times more likely to develop a peptic ulcer than persons of other blood groups. Persons with blood group A have higher incidence of gastric carcinoma.

#### **c. Liver Function Tests**

They are done in suspected cases of gastric cancer to assess liver involvement. Abnormalities in liver function tests predict unresectability in 76-89% of instances and if there is significant elevation in 3 or 4 parameters, radical resection is contraindicated.

#### **d. Serum electrolytes**

It is often overlooked. It may show hypochloremic, hypokalemic, hyponatremic metabolic alkalosis.

**Urine examination.**

Initially urine is alkaline which later becomes acidic with large amounts of  $\text{Na}^+$  and  $\text{H}^+$ . Urine is concentrated with high specific gravity. albumin, sugar and microscopy are done to rule out associated conditions. Bile salts and bile pigments are done in cases of carcinomas and duodenal tuberculosis.

**Occult Blood in Stool**

Occult blood in stool is detected by Benzidine test. An emulsion of a little of the faeces is heated to boil in a test tube and cooled immediately in cold running water to destroy any enzymes present. In another test tube, 1 ml of glacial acetic acid and a pinch of Benzidine powder is taken and the mixture is heated and cooled. This is then added to the emulsified & heat treated faeces. 2 ml of hydrogen peroxide is then added to it. The solution will turn blue or green if blood is present. This test is not diagnostic of gastric malignancy as bleeding anywhere in the gut can give a positive test. The patient is asked to avoid meat and iron preparations for atleast 3 days prior to the test, as these can give rise to false positive tests. Anaemia is a constant feature in patients with gastric outlet obstruction and carcinoma stomach.

**Gastric Secretion Tests****Measurement of Gastric Emptying**

A variety of methods have been used to measure gastric emptying many of which give only limited information.

## **1) Radiological methods**

The addition of radio opaque markers to a liquid or solid meal has been used to study gastric emptying. Interpretation of the results, however, is limited as only two parameters can be measured: the time between complete ingestion of the meal and the start of gastric emptying and the time to complete gastric emptying. Barium is much heavier than normal food, is extremely viscous, and adheres to the gastric mucosa. Methods using barium incorporated in meals, and enteric coated barium granules have been only partially successful. The dose of radiation that may be given during these examinations may be as high as 5 rads.

## **2) External scanning techniques**

Griffin et al, in 1966, first measured the rate of gastric emptying using the radio – isotope chromium – 51 chloride incorporated in a meal. Counts from the meal were detected by a scanning detector, measuring activity over the stomach area. The number of counts from the stomach area was determined for each scan and an exponential pattern of emptying assumed. This method was found to be deficient as it did not take early emptying into account. As a result of the deficiencies with scintiscanning, Harvey et al (1970) advocated the use of the gamma camera, which counts the whole stomach area simultaneously, for measuring emptying. The limitation of this method is that it is difficult to determine what fraction of the isotope remains in the solid phase. Solid components of the meal which differ in shape, consistency and other physical properties are emptied from the stomach at different rates. Because of

fluctuations in the post prandial intragastric secretions, gamma camera monitoring can estimate only the amount of meal remaining in the stomach, which is the most important parameter. Gastric emptying of solids is more reliable because it is largely independent of the volume of intragastric liquid. The large, low lying stomach found in most cases of outlet obstruction is extensively overlapped by small bowel. Thus upper small bowel contents labelled with the radio isotope, will cause an artificially high gamma count from the stomach area producing an artificially high estimate of the amount of meal remaining in the stomach.

### **3) Nasogastric intubation techniques**

**a) Saline load test:** Originally introduced by Goldstein and Boyle in 1965, it involves nasogastric intubation and instillation of a known volume of isotonic saline (750 ml). The stomach is aspirated after 30 minutes and the residual volume recorded. In a normal individual less than 200 ml is aspirated, while in gastric outlet obstruction the aspirate is above 400 ml. The disadvantage is that since it only measured emptying of isotonic saline, this test is insensitive and offers no information on how the stomach handles solids or commonly ingested liquids.

**b) The fractional test meal:** This test was originally designed to study gastric secretion but may also be used to measure emptying. A meal of gruel is given and gastric aspirations are taken at 15 minute intervals till a starch test reveals no remaining gruel in the stomach. The end point of the test is difficult

to determine as the starch test is very sensitive and tiny amounts of gruel in the stomach will give a positive result.

**c) The serial test meal:** This test was devised because of the limitations of the fractional test meal. A known volume of liquid containing a non-absorbable marker (phenol red) is given and the entire gastric contents are aspirated after a specific time interval. The test is repeated on several days with the meal being removed after different time intervals. By estimating the amount and concentration of the marker in each sample it is possible to determine the rate of gastric emptying. The disadvantages of this test are that it takes several days to complete and requires repeated nasogastric intubation and so is very unpopular with patients. However, it is a reliable and reproducible test.

#### **4) The double sampling dye dilution test**

In order to overcome the repeated intubations and the time disadvantages of the serial test meal, George (1968) described a method using double sampling of the stomach contents. This enabled the complete course of gastric emptying to be studied during a single examination. After drinking a liquid meal a small amount of stomach contents is aspirated via a nasogastric tube. Marker of known volume and concentration is then added to the stomach contents and mixed for one minute by repeated aspiration and replacement of the stomach contents. Second sample is then aspirated and the concentration of the marker in the two samples determined. A formula is then used to calculate the volume of the stomach contents at the time the first amount was withdrawn

from the stomach. The procedure is repeated at regular intervals, and a graph plotted of stomach volume against time. This test measures total stomach volume, which includes the remaining original meal, swallowed saliva, gastric secretion and any duodenal reflux. The main source of error in the double dye dilution technique is in the mixing of the marker with the stomach contents. Uniform mixing is difficult to achieve and the gastric contents are not normally homogenous.

### **5) Real – time ultrasound**

The use of real –time ultrasound is a method of measuring gastric emptying (Bateman and Whittingham – 1982). Short pulses of ultrasound pass through liquid and are partially deflected by solid interfaces, producing echoes. The time taken for an echo to return to sensor transducer on the skin is determined by the depth of the reflecting surface. A three – dimensional image of the stomach can be produced by a series of cross – sectional images at regular 1 cm intervals. The volume of the stomach can be obtained from measurements of the areas of cross sectional images.

Ultrasound is considered to be safe and this technique is non-invasive and readily reproducible. This method is limited to liquid meals, and gastric overlap of gas or liquid containing small bowel may limit its use. Further evaluation is required to assess its true potential in patients.

## Gastric Secretion Tests

The gastric secretion tests can be done as either outpatient or in-patient procedure, but must be performed by an experienced doctor, nurse or technician. Tests are performed early in the morning. The patient should have had nothing to eat or drink from the previous night and any drugs affecting gastric secretion, should not have been taken for the previous 24 hours. A large bore nasogastric tube is passed into the stomach. The stomach is emptied of its contents by repeated suction and one 60 minute sample of gastric juice is collected and labelled to estimate basal secretion.

**PENTAGASTRIN TEST.** — The optimum dose of pentagastrin is 6 microgram per Kg body weight. It is mostly injected I. M. 15 minutes samples are collected during the next one hour. The term maximum acid output (MAO) is generally used as the maximum acid output in the whole 60 minutes after injection of pentagastrin is expressed as m.mol/hr. This test not only has a diagnostic importance but also helps in assessing treatment of the patient e.g. when the acid status is low operation like vagotomy and drainage procedure is enough; whereas in case of high acid status individuals operation like vagotomy and antrectomy or partial gastrectomy should be called for. In gastric carcinoma MAO is very low.

**KAY'S AUGMENTED HISTAMINE TEST.** — This test determines the total mass of oxyntic cells in the stomach. At first the fasting stomach contents are collected. Mepyramine maleate is given intramuscularly at the dose of 100 mg



to nullify the side effects of histamine except its stimulation of gastric acid. About 30 minutes later histamine acid phosphate at the dose of 0.04 mg per Kg body weight is injected subcutaneously. The gastric acid secretion is collected during the next one hour. The average HCl response in mEq free acid per hour is as follows: a gastric ulcer - 15; duodenal ulcer - 30 to 40; anastomotic ulcer - 30 to 35.

**HOLLANDER'S INSULIN TEST** - This is based on the fact that hypoglycemia, caused by insulin, induces direct vagal stimulation on the parietal cell mass. Insulin given to a patient who has had a vagotomy performed should result in no increase in acid production. This test is of more value to assess the completeness of vagotomy in the postoperative period. After the fasting stomach contents are aspirated, insulin in the dose of 0.2 units per Kg. of body weight is injected intravenously. 2 ml of venous blood is taken just before the introduction of insulin for estimation of sugar. Eight 15- minutes aspirates are collected and labeled. 2 ml of venous blood is taken at 30 and 45 minutes after introduction of insulin to estimate the level of blood sugar. It must be remembered that patient's comments and appearance should be noted. Dryness and slight impairment of the level of consciousness should immediately rouse the suspicion of imminent hypoglycemic coma. Dextrose (50 percent) in the dose of 50ml should be always kept available during the test and injected immediately intravenously should the said condition arise. A fall in the blood sugar level below 45mg per 100 ml will lead to hypersecretion of acid.

Maximum acid output is expected at that time. A rise in concentration of 20 m. mol per liter above the basal level in the first hour suggests incomplete vagotomy. It must be remembered that a high acid concentration in basal secretion in the range of more than 20 m. mol free acid per hour is indicative of Zollinger-Ellison syndrome.

### **Sham Feeding Test**

This test was developed as a safer alternative to the insulin test. In this test the patient chews and spits out a tasty meal and the mouth is washed out with a small quantity of water to ensure no food particle is swallowed. The chew, spit and rinse sequence is repeated for several cycles for a total of 10 minutes during which gastric secretion is continuously aspirated. Additional aspiration for another five 10- minute periods are done. In this test the gastric secretion is stimulated in a manner similar to that of the cephalic phase of gastric secretion and is mediated by the vagi.

### **Radiological Investigations:**

**(1) Pain X-ray Abdomen:** Plain X-ray abdomen is useful in detecting foreign bodies in the stomach. In cases of Trichobezoar, it may reveal an intra-gastric foreign body outlined by surrounding gas giving a mottled appearance.

**(2) Chest X-ray:** Chest X-ray is done to rule out pulmonary secondaries and also to assess preoperatively the lung function

**(3) Barium Meal Examination:** Barium meal examination is a common investigation being done to diagnose gastric outlet obstruction. The test is done

in empty stomach after overnight fasting. As the barium enters the stomach it sinks through a layer of fluid and comes to rest at the bottom of the greater curvature to produce a saucer like appearance indicating the presence of excessive fasting gastric juice. The X-ray film taken in erect posture shows three layers – air, gastric juice and barium separated from each other by two fluid levels. Giant peristaltic waves are seen which indent the greater and lesser curvature during the compensated phase of pyloric obstruction and emptying of the stomach is seen upto three hours. But a considerable amount of barium (greater than 50%) is seen remaining in the stomach even after 4-6 hours. In the decompensated phase of pyloric obstruction, the stomach is seen as a large atonic bag and is characteristic of benign obstruction.

Pylorospasm can be eliminated by intravenous injection of propantheline bromide which results in normal emptying.

When the obstruction is beyond the pylorus, abnormal dilatability of the pyloric canal is seen. The canal may dilate upto 2.5cms in width and contract down to its usual size, which is a sign of obstruction in the first part of the duodenum. When the scarring of the duodenum is central, it may be seen as irregular, star – shaped duodenal cap. When the scarring is eccentric, there is an additional formation of prestenotic diverticulum.

Ulcerative growths show an irregular crater with rolled edges of the growth forming a half shadow around the crater (Carman's sign). When the obstruction is situated at the gastric antrum, malignancy is the most likely

diagnosis. The antral wall appears rigid with irregular contour and lack of peristalsis. The antrum may give a tapered appearance and there may be a persistent irregular filling defect caused by malignant ulcer.

Adult hypertrophic pyloric stenosis is recognized by the bulbous intrusion into the base of the duodenal cap.

Pancreatic carcinoma may show widened C-loop or inverted '3' sign. Heterotopic pancreatic tissue may sometimes be demonstrated by the presence of a thin streak of barium which communicated with the pyloric lumen and represents the duct of the ectopic pancreas.

Early malignant lesions in the stomach are best demonstrated by double – contrast barium study. The stomach is distended with gas while a thin coating of barium enables its inner surface to be visualized. On distension of the stomach, the gastric mucosa is put under slight tension and lesions causing lack of distensibility results in a clearly visible series of converging folds. With this technique, small lesions and slight irregularities of the mucosa can be identified. This technique is particularly useful in detecting early gastric cancer and is thought by many, to be superior to endoscopy in this respect.

### **Upper Gastro – Intestinal Endoscopy**

With the advent of flexible fibre optic endoscope, upper G-I endoscopy has become the investigation of choice in diagnosing lesions in the upper gastro – intestinal tract. The patient is kept nil by mouth 4 – 6 hours prior to the procedure. The procedure is usually done under sedation. The patient is

intubated under direct vision from the left side with the neck flexed and mouth guard in-situ. Air is insufflated into the stomach when the endoscope is at the level of the oesophagogastric junction. Gastric carcinoma may appear as a cauliflower – like protuberance, an ulcer or as an infiltration of the sub-mucosa. Biopsy from the site of lesion is taken for confirming the diagnosis. This is one of the main advantages endoscopy has over barium – meal examination. However, to diagnose early gastric cancer through an endoscope, it is still difficult and it is no better than conventional radiography in this regard.

The endoscope can be used as a therapeutic tool in various situations.

1. To retrieve foreign bodies which are impacted in the pylorus causing obstruction.
2. To dilate the stenosed portion at the gastric outlet, by balloon dilatation
3. To place metallic stents in cases of inoperable gastric carcinoma, to relieve the obstruction.
4. Small pedunculated polyp can be excised through the endoscope.

### **Abdominal Ultrasound**

The use of ultrasound in detecting gastric lesions is very limited, as the gas present in the stomach reflects all the incident beam of sound. In cases of gastric malignancy, ultrasound is helpful in detecting regional lymph node enlargement, hepatic metastasis and ascites.

Endoluminal ultrasound and laparoscopic ultrasound are probably the most sensitive techniques available in the preoperative staging of gastric cancer.

In endoluminal ultrasound, the transducer is usually attached to the distal tip of the instrument. However, devices have been developed that may be passed down the biopsy channel, albeit with poorer image quality. Five layers of the gastric wall may be identified on endoluminal ultrasound and the depth of invasion of a tumour can be assessed with exquisite accuracy [90% accuracy for the 'T' tumour component of the staging]. Enlarged lymph nodes can also be identified and the technique's accuracy in this situation is about 80%. Finally, it may be possible to identify liver metastases not seen on axial imaging. Laparoscopic ultrasound is also very sensitive and is one of the most sensitive methods of detecting liver metastases from gastric cancer.

### **CECT Abdomen**

CECT Abdomen is useful in demonstrating the intramural, extra serosal and metastatic spread of malignancy. It shows tumour size, extent, infiltration, lymphnode status, secondaries, ascites and operability. In this way it helps to avoid unnecessary surgery in patients with incurable lesions. The detection of small liver metastases is improving although, in general terms, metastases from gastric cancer are less easy to detect using CT than those, for instance, from colorectal cancer. This is because metastases from gastric cancer may be of the same density as liver and may not handle the intravenous contrast any differently.

The accuracy of CT assessment of tumour location and T stage can be enhanced over that of conventional helical CT by use of water as an oral contrast agent, the so-called helical hydro-CT.

### **Positron Emission Tomography**

Whole body 2-[18F]-fluoro-2-deoxyglucose (FDG) positron emission tomography (PET) is being applied increasingly in the evaluation of gastrointestinal malignancies. Positron emission tomography (PET) is a functional imaging technique that relies on the uptake of a tracer, in most cases by metabolically active tumour tissue. To be of value, anatomical and functional information need to be linked, hence CT/PET is now used universally.

### **Laparoscopy**

Staging laparoscopy has become an accepted part of the pre-treatment staging evaluation of patients felt to have localized gastric cancer after initial helical CT assessment. The rationale for laparoscopic staging is based on the fact that the sensitivity of CT for detection of extra gastric disease declines with the size of metastases. Indeed, current CT techniques cannot consistently identify low volume macroscopic metastases that are 5 mm or less in size. Laparoscopy allows for direct inspection of the peritoneal and visceral surfaces for detection of CT-occult small volume metastases. Staging laparoscopy also allows for assessment of peritoneal cytology and intra peritoneal evaluation with adjunctive diagnostic techniques such as laparoscopic ultrasound. Patients

who are found to have occult metastatic disease at laparoscopy are considered incurable, and the use of laparoscopy allows them to avoid laparotomy

### **Tumor Markers**

The carcinoembryonic antigen (CEA) level is elevated in approximately one third of patients with primary gastric cancer. The sensitivity of CEA as a marker of gastric cancer is low, but when the CEA level is elevated, it does generally correlate with stage. Combining CEA with other markers, such as the sialylated Lewis antigens CA 19-9 or CA 50, can increase sensitivity compared with CEA alone.

### **TREATMENT**

Careful preparation is important as the surgery in these cases otherwise carries a significant mortality. The presence of dehydration signs suggests an approximate fluid deficit of 4 litres and about 20gms of NaCl. Provision of sodium allows excretion of alkaline urine, so that alkalosis becomes correctable. The hypokalemic, hypochloremic acidosis is best corrected by administering normal saline containing potassium chloride. When hypokalemia is severe and requires more than 20meq of KCl per hour needs to be infused; cardiac rhythm should be monitored continuously. Rarely central venous monitoring becomes necessary, but if it is, the administration of large quantities of KCl centrally must be avoided. In rare cases with severe alkalosis, IV infusion of isotonic solution of dilute HCl may be required. The general condition should be



improved by treatment of dental caries, chest physiotherapy, and vitamin supplementation. Nutritional support may be provided by a central line or internally via laparoscopically placed jejunostomy feeding tube. Success is indicated by clinical improvement in the state of hydration, increased urine output; fall in blood urea and hematocrit and normal electrolyte concentration.

## **Gastric outlet obstruction due to chronic peptic ulcer**

### **A. Endoscopic Intervention**

About 85% of gastric outlet obstruction cases are amenable to dilatation and 80% of these had immediate relief from the symptoms. Only 40% of these sustained improvement after three months.

#### **1. Through the scope (TTS) balloon dilatation**

A well lubricated balloon is passed through the biopsy channel. Balloon is inflated to maximum pressure in stenosed area with water or diluted contrast medium using pressure gauze. Pressure is maintained for one minute and repeated for 3-4times.

#### **2. Over the wire balloon dilatation**

A guide wire is advanced far enough in the stricture area. Dilatation is done under fluoroscopic guidance using dilute contrast medium. Pyloric ring and duodenal bulb are examined endoscopically.

Patient who have an identifiable cause (e.g., *H. pylori* infection) that could be treated, have good long-term results with endoscopic dilation, with a median of five dilations required, but no subsequent surgical therapy.

Boylan et al found that young age, long duration of symptoms needing more than one session of EBD and continuous use of NSAIDs were associated with adverse outcomes and predicted the need for multiple dilations and surgery. DiSario et al reported a longer length of the narrowed segment was a poor prognostic sign.

## **B. Surgical treatment**

It involves the relief of obstruction and antiulcer treatment. The choice of operation is between vagotomy and antrectomy and vagotomy and drainage. The recommended operation is vagotomy and antrectomy with insertion of a feeding jejunostomy tube to provide postoperative enteral nutritional support. In cases of severe inflammation that preclude safe resection of the duodenum, vagotomy and gastrojejunostomy are recommended. Truncal vagotomy and gastroenterostomy conserves the gastric reservoir and can be done with a lower risk. Truncal vagotomy and gastroenterostomy has operative mortality of less than 1% and 2 - 7% ulcer recurrence rate.

### **Modified Visick grading system for peptic ulcer surgeries**

Grade 1- Perfect result, an asymptomatic patient

Grade 2 - Mild, intermittent symptoms that are easily controlled by diet.

Grade 3 - Moderate symptoms without substantial interference with lifestyle.

Grade 4 - Unsatisfactory outcome and includes all patients with recurrent ulcer.

A newer approach is to perform a highly selective vagotomy(HSV) or proximal selective vagotomy with either a pyloroplasty or gastrojejunostomy. This preserves antral mill and reduces the incidence of dumping, gastric stasis and bile reflux gastritis. It also produces superior nutritional status and iron is better absorbed as duodenum is not bypassed. It has mortality rate of less than 0.2% and recurrence rate of 2-10%. The problems associated are dysphagia due to dissection of last 7cm of esophagus and damage to nerve of Latarjet.

The simplest procedure to perform through minimally invasive technique is truncal vagotomy and gastrojejunostomy. Gastrojejunostomy can be accomplished through endoscopic stapling device.

### **Truncal vagotomy**

The main indication is gastric outlet obstruction with a long-standing history of ulcer symptoms or bleeding or perforation. Because the vagal nerves are conductors of motor impulses to the stomach, denervation of the antropyloroduodenal segment results in gastric stasis in a substantial proportion of patients on whom truncal vagotomy alone is performed. Hence it should be performed in conjunction with some drainage procedure. The complications include

- Intra-operative: injury to distal esophagus, splenic vessels, diaphragm, liver.
- Early post-operative: delayed emptying, dysphagia.

- Late post-operative: diarrhoea, reflux esophagitis and cholelithiasis.
- Post – vagotomy diarrhoea can be the most devastating symptom to afflict the patients having peptic ulcer surgery. The precise aetiology of the problem is uncertain. It is partly related to rapid gastric emptying.

### **Highly selective vagotomy**

Highly selective vagotomy can be divided into 4 phases

- (1) Exposure and gastric mobilization
- (2) Dissection of the anterior leaf of the lesser omentum
- (3) Dissection of the posterior leaf of the lesser omentum
- (4) Dissection of vagal fibers travelling to the stomach along the distal esophagus.

HSV can be used in combination with finger or endoscopic balloon dilatation in cases of pyloric outlet obstruction.

### **Drainage procedures**

The options available are

#### **1.Pyloric dilatation**

a. **Open method:** perform a small gastrostomy, approximately 3-4cm in length proximal to pylorus. It is widened using finger and then is sutured in single layer of 3-0 interrupted silk sutures.

b. **Laparoscopic method:** using a balloon, 15mm in length, which may be positioned endoscopically and inflated to 45 psi for 10 minutes.

## **2. Pyloromyotomy**

The incision is made to score the anterior surface of the stomach from 1-2 cm proximal to 1 cm distal to the pyloric ring. The seromuscular layer is dissected without disrupting the mucosa. The separation of pyloric muscles is accomplished mainly with a fine-tip haemostat and the knife. An omental patch is used to cover the dissected area. It can also be done via laparoscopy along with truncal vagotomy.

## **3. Pyloroplasty:-**

Can be done in two ways.

### **a. Heineke-Mikulicz procedure**

The most expeditiously performed pyloroplasty is the Heineke-Mikulicz procedure. It is not always necessary to perform a Kocher manoeuvre; however, duodenal mobilization is usually helpful in relieving any tension on the intended suture line. Incision is made on the anterior surface in a longitudinal direction, using electrocautery, from 2 cm distal to the pyloric muscle to 3 cm proximal to the pylorus. The closure of the pyloroplasty is performed vertically, in order to minimize narrowing of the lumen.

### **b. Finney pyloroplasty**

The Finney pyloroplasty can be used when scarring has involved the pylorus and duodenal bulb and would not permit a tension-free, patulous Heineke-Mikulicz pyloroplasty. The Finney pyloroplasty is in essence a side-to-side gastroduodenostomy. The Kocher manoeuvre is performed, carrying the

mobilization distally. Complete mobility of the duodenum and freedom from surrounding adhesions are essential to this operation. An inverted U-shaped incision is made into the lumens of the stomach and duodenum and a gastroduodenostomy completed.

#### **4. Gastroenterostomy**

A posterior retrocolic, isoperistaltic, no loop and no tension, vertical gastrojejunostomy is usually made. The opening in the stomach is made vertical (Moynihan) for easy evacuation, though oblique (Mayo) or horizontal (Kocher) opening does not make much difference in emptying.

#### **Gastric outlet obstruction due to gastroduodenal tuberculosis**

Antitubercular therapy is only that is required in partial obstruction. Gastrojejunostomy is advised for complete obstruction while distal partial gastrectomy if multiple tubercular ulcers are present.

#### **Gastric outlet obstruction due to corrosives**

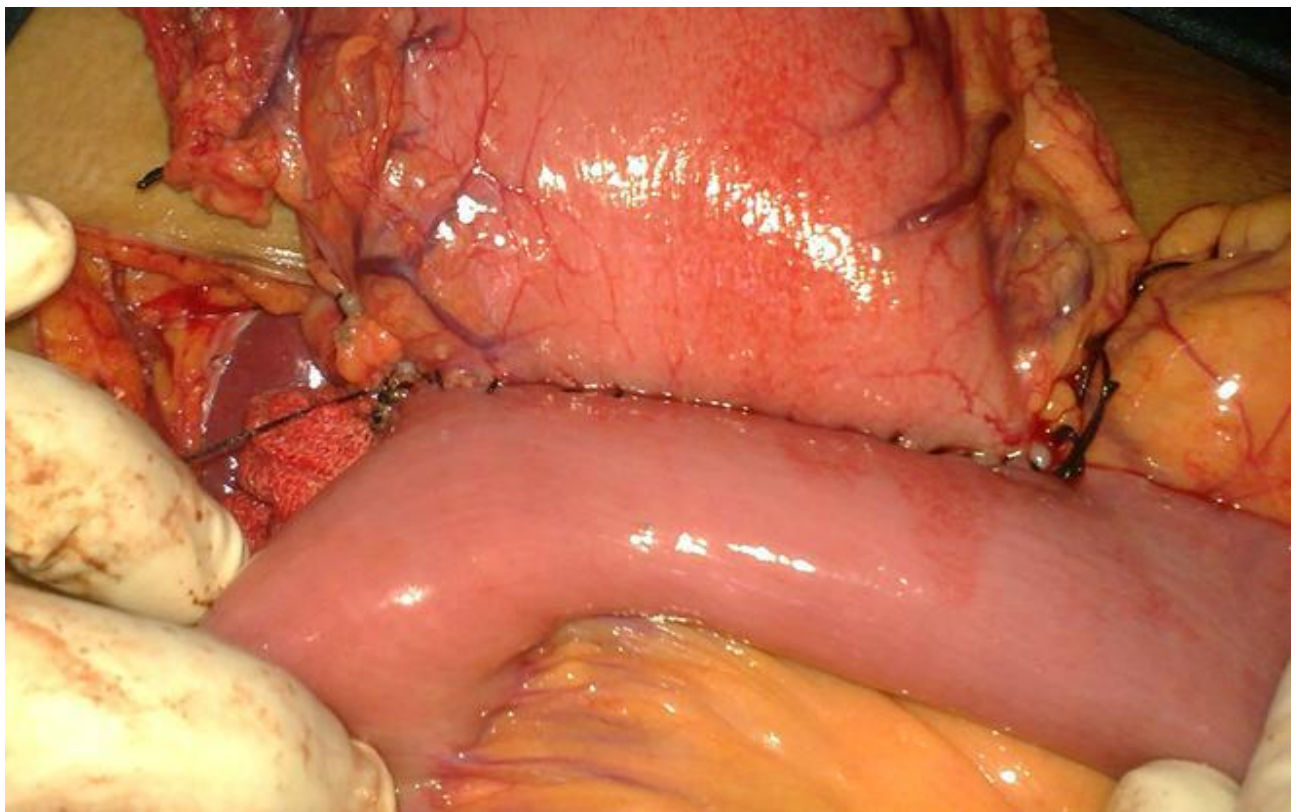
Gastrojejunostomy is preferred. Partial gastrectomy can also be done.

#### **Gastric outlet obstruction due to post-operative scarring**

Adhesiolysis is enough except when an organic stenosis is present where gastrojejunostomy or partial gastrectomy is done.

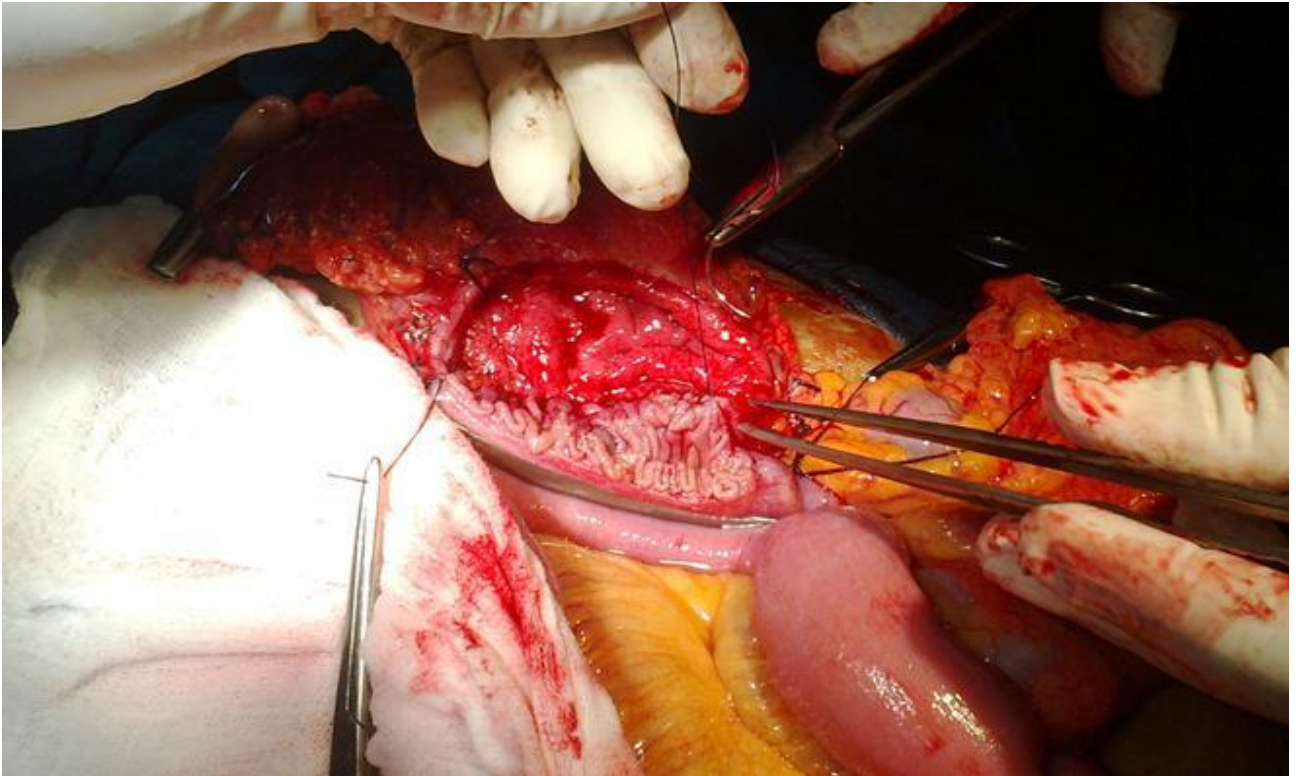
### **Gastric outlet obstruction due to acute/chronic pancreatitis or pseudocyst**

Surgical treatment is required in patients who continue to have symptoms even after 3-4 weeks of medical line of management to reduce inflammation and edema of duodenal wall. Obstruction may be relieved by inflammatory adhesions around the duodenum or drainage of pseudocyst. Obstruction due to chronic pancreatitis is treated by vagotomy or gastro-jejunostomy or resection. Gastrojejunostomy is enough to relieve duodenal obstruction. Vagotomy is added if a patient has a past history of acid peptic disease.

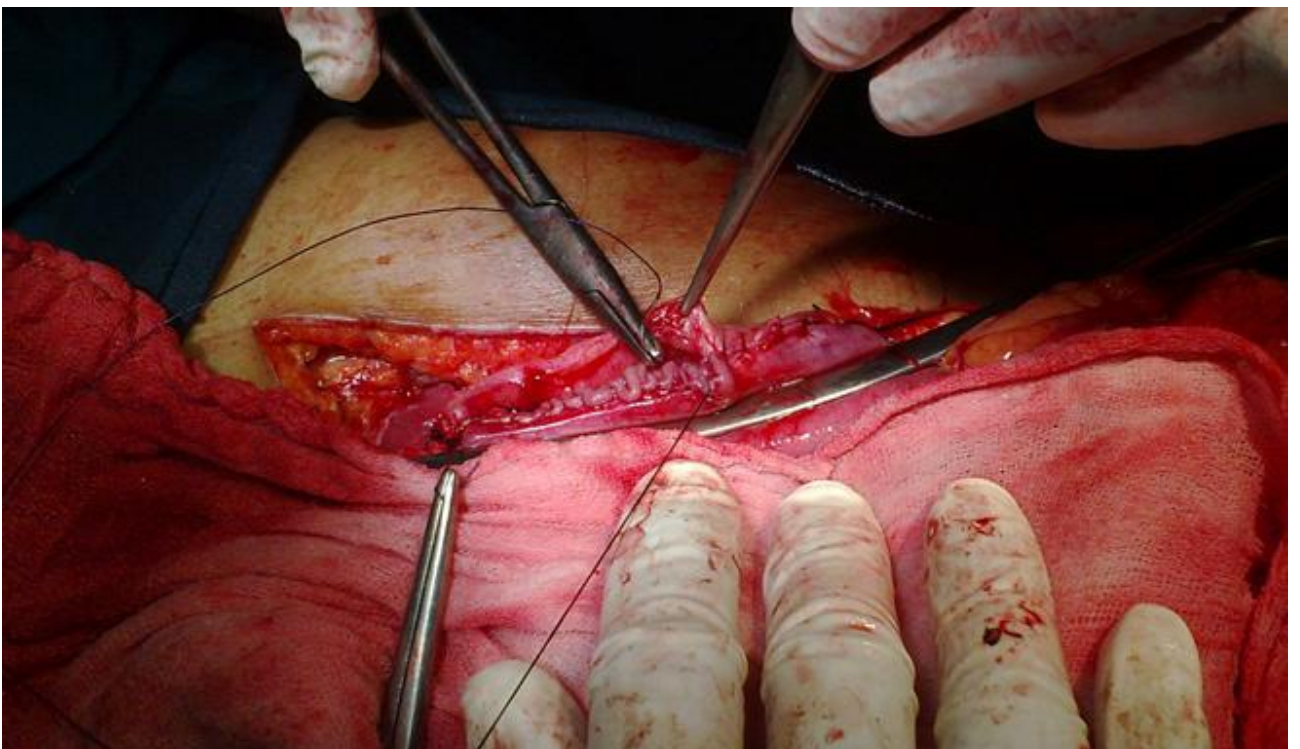


**FIGURE 5. THE FIRST LAYER OF GASTRO-JEJUNOSTOMY.**



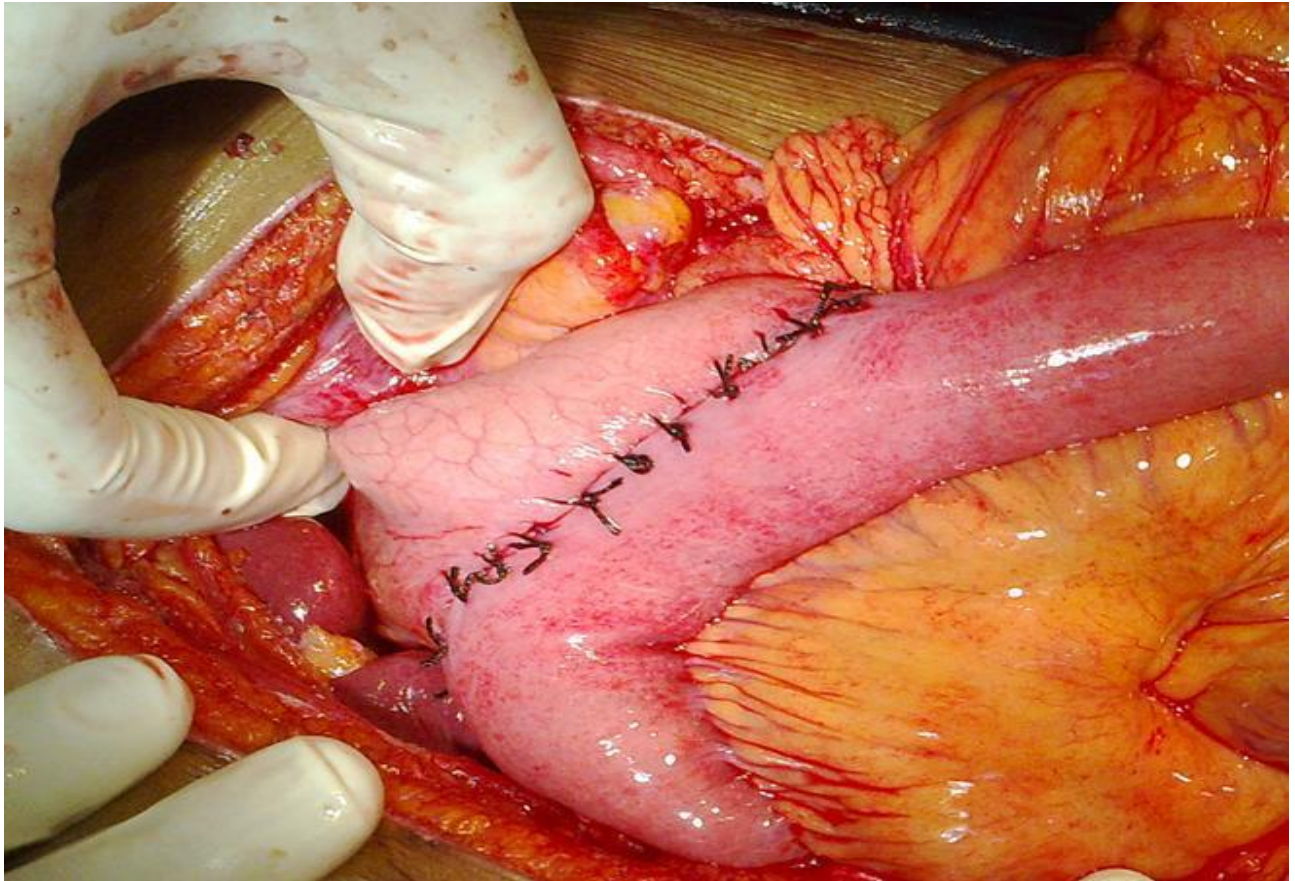


**FIGURE 6. THE SECOND LAYER OF GASTRO-JEJUNOSTOMY.**



**FIGURE 7. THE THIRD LAYER OF GASTRO-JEJUNOSTOMY.**





**FIGURE 8. THE FOURTH LAYER OF GASTRO-JEJUNOSTOMY.**

### **Gastric outlet obstruction due to carcinoma stomach**

Complete resection of the gastric tumour with a wide margin of normal stomach remains the standard of care for resection with curative intent. The extent of resection depends on the location of the tumour in the stomach and size of the tumour.

The signs of inoperability are:

### **Pre-operative signs**

1. Malignant ascitis
2. Jaundice
3. Gross cachexia

4. Secondaries in rectovesical or rectouterine pouch (Blummer shelf)
5. Sister Mary Joseph's nodules
6. Krukenburg tumour
7. Secondaries in left supraclavicular lymphnodes

### **Per-operative signs**

1. >H2
2. >P2
3. >N4
4. Omental deposits

For cancers of the distal stomach, including the body and antrum, a distal gastrectomy is the appropriate operation. The proximal stomach is transected at the level of the incisura at a margin of at least 6 cm, because studies have documented tumour spread as far as 5 cm laterally from the primary tumour. Frozen section analysis should be performed prior to reconstruction. The distal margin is the proximal duodenum. The possibility of recurrence in the tumour bed (duodenal suture line and surface of the pancreas) suggest a Billroth II reconstruction rather than a Billroth I, which will result in less risk of gastric outlet obstruction secondary to tumour recurrence.

For early gastric cancer with limited penetration of the gastric wall and no evidence of lymph node metastases, purely endoscopic mucosal resection can be carried out. The general guidelines for endoscopic resection of early gastric cancer are as follows:

- (1) Tumour limited to the mucosa;
- (2) No lymphovascular invasion;
- (3) Tumour smaller than 2 cm; and
- (4) No ulceration.

A finding of any of these on initial biopsy or during endoscopic resection is an indication for gastrectomy with lymph node dissection.

### **Lymph Node Dissection**

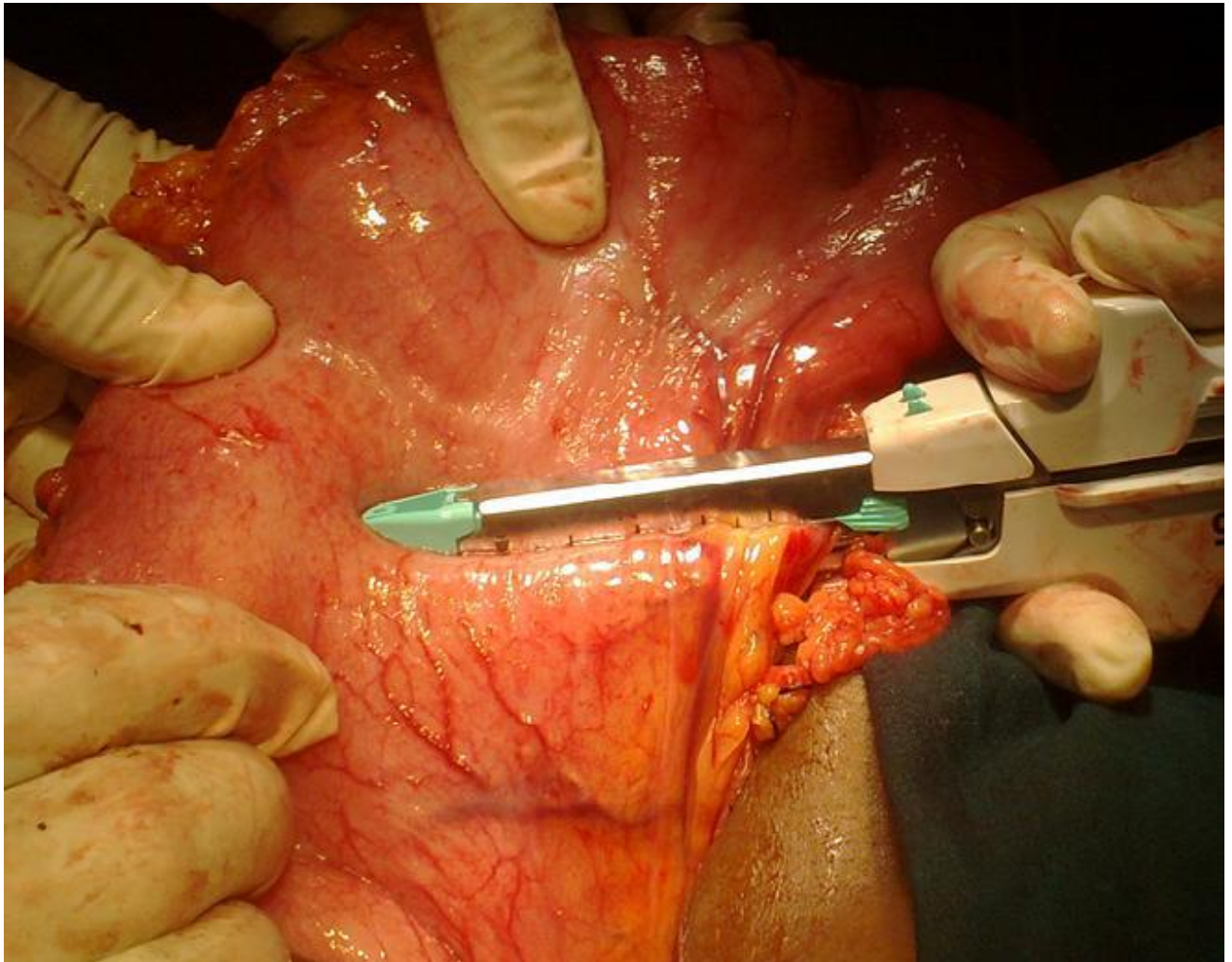
At least 15 lymph nodes have to be removed for adequate staging purposes.

D1 lymphadenectomy - clearance of only perigastric nodes

D2 lymphadenectomy - clearance of the celiac axis, with or without splenectomy

D3 lymphadenectomy - clearance of the celiac axis and periaortic nodes.

The Japanese have shown increased survival in patients undergoing a D2 dissection, with no increased or minimal increase in morbidity.

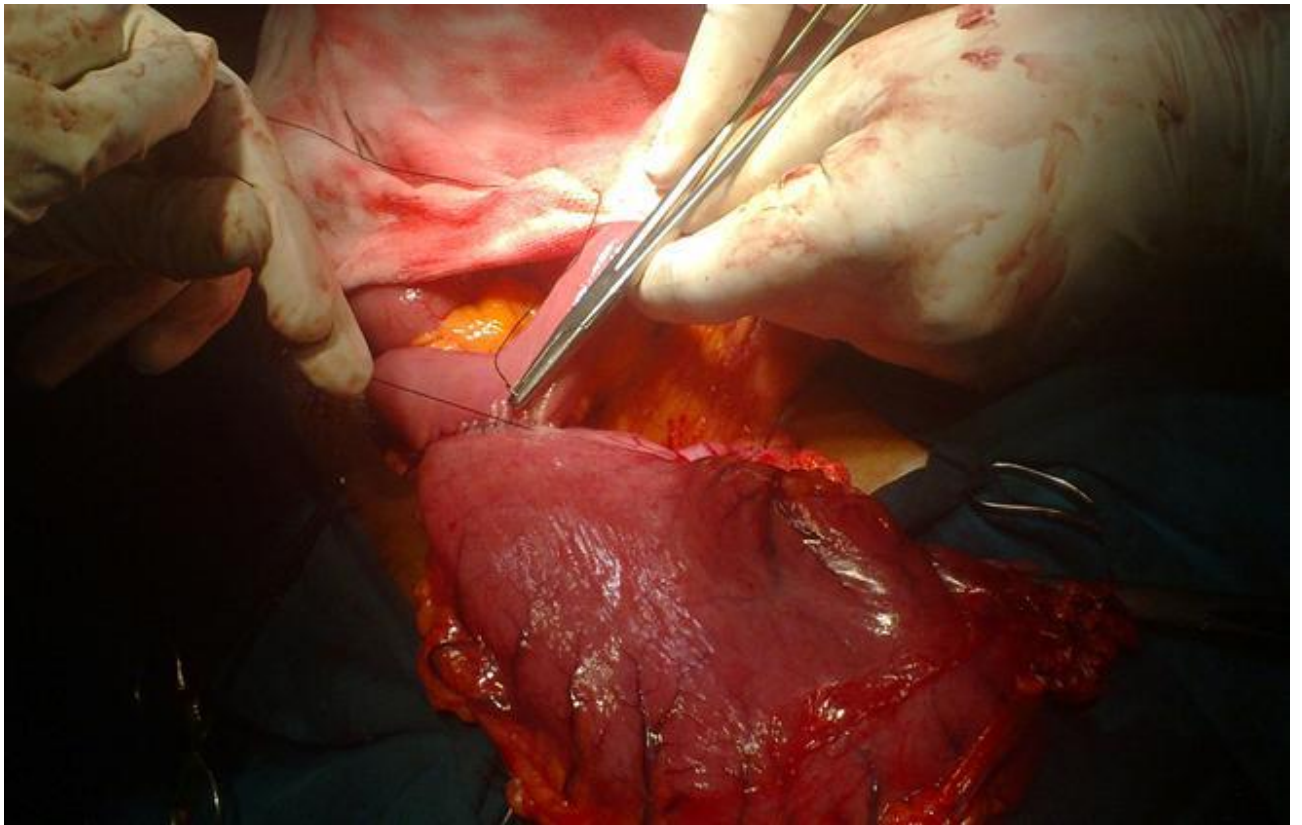


**FIGURE 9. SUBTOTAL GASTRECTOMY. LINEAR CUTTER BEING USED TO SECTION AND STAPLE THE STOMACH, FROM THE LESSER CURVE SIDE.**

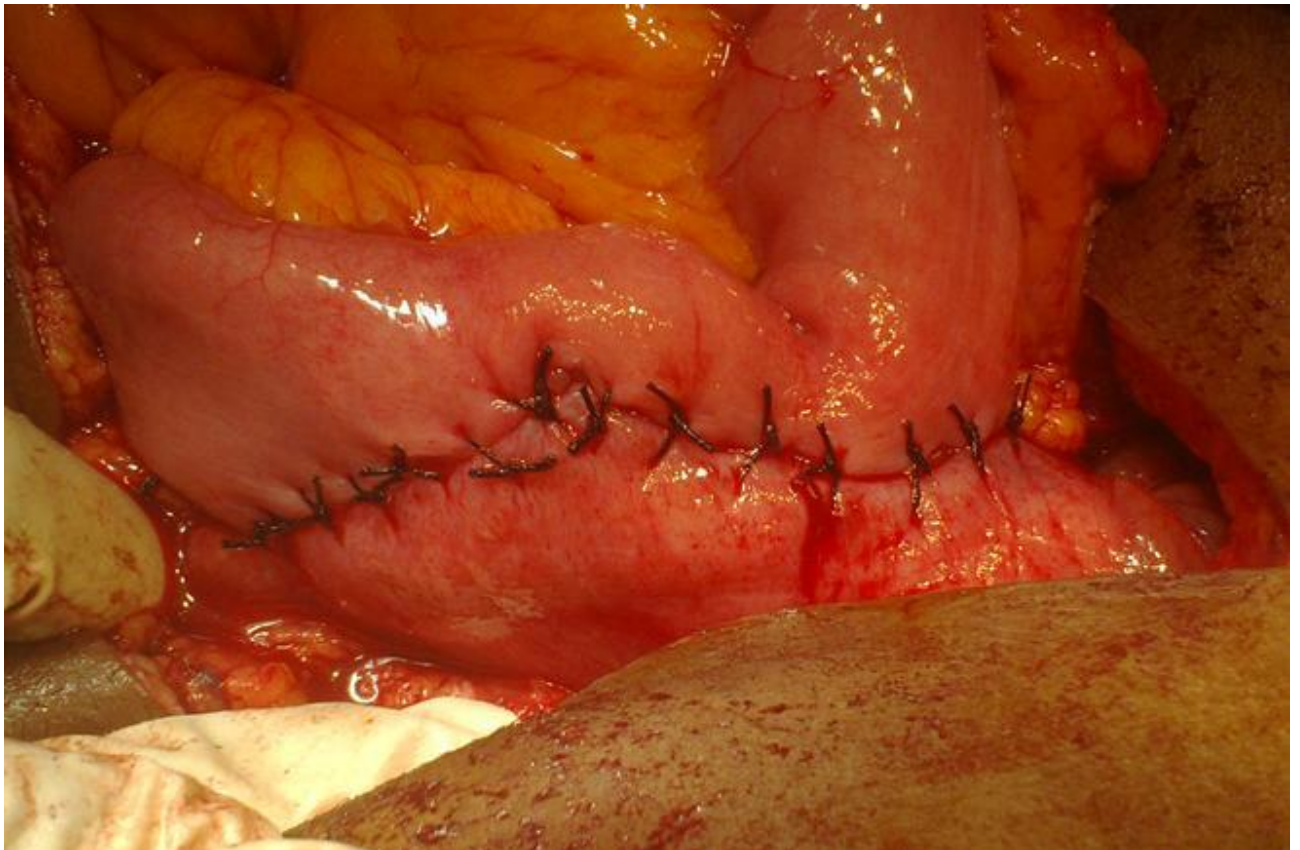




**FIGURE 10. SUBTOTAL GASTRECTOMY IN PROGRESS. PARTIALLY STAPLED STOMACH FROM THE LESSER CURVE SIDE.**



**FIGURE 11. HANDSEWN GASTRO-JEJUNOSTOMY IN PROGRESS FOLLOWING PARTIALLY STAPLED STOMACH FROM THE LESSER CURVE SIDE.**



**FIGURE 12. SUBTOTAL GASTRECTOMY WITH GASTRO-JEJUNOSTOMY COMPLETE, COMBINATION OF STAPLED AND HANDSEWN TECHNIQUE.**

## **Advanced Gastric Cancer**

If the growth is not fixed, palliative resection may be done to improve the quality of life and to enhance patient response to chemotherapy. If the palliative resection is not possible, anterior gastrojejunostomy is done to relieve the obstruction.

If the patient is not in a position to undergo any surgical procedure, metallic stenting to overcome the obstruction can be tried as an alternative. The stenting can be done through the endoscope or by a mini-laparotomy under local anaesthesia.

### **Adjuvant therapy**

For advanced cancers (deeply penetrating T2 or T3 tumours, patients with lymph node metastasis) systemic therapy is advised as there is high propensity for systemic failure with or without local recurrence. Commonly used combination chemotherapy is EAP (Etoposide + Adriamycin + Cisplatin) and FAM (5-FU + Adriamycin + Methotrexate).

### **Intraperitoneal Chemotherapy**

The rationale for the use of intraperitoneal treatment is based on the pharmacokinetic observation that drug concentrations within the peritoneal cavity after intraperitoneal administration are much higher than those achievable intravenously or orally. It can be given either as heated solutions (continuous hyperthermic peritoneal perfusion, or CHPP) or nonhyperthermic treatment given either immediately or instituted within several days of



resection. CHPP has the advantage of synergism from CT and cytotoxic hyperthermia. Either a fluorinated pyrimidine such as fluorouracil or floxuridine, or mitomycin C with or without other agents can be given. Cisplatin can also be added.

### **Immunotherapy**

Protein-bound polysaccharide (PSK) or a *Streptococcus pyogenes* preparation, OK432 can be used in combination with various combination CTs.

### **Perioperative (Neoadjuvant) Chemotherapy**

Perioperative chemotherapy to be given in patients who have locally advanced tumours at diagnosis (T3 or T4, or obvious lymph node involvement). Such patients are not only at substantial risk for distant metastasis, but local extent of the tumour may make an R0 resection difficult. The two goals of perioperative treatment are to reduce the stage of the primary tumour to increase the likelihood that a R0 resection can be performed, and to begin at an early time to treat micrometastatic disease.

### **Radiation therapy**

In palliation, radiation therapy can be used for bleeding or controlling pain secondary to local tumour infiltration. Stomach is radiosensitive and ulcerates and bleeds at doses higher than 50 Gy. Intra-operative RT can be given to tumour bed. In chemoradiation it is usually combined with 5-FU.

## **Targeted Therapy**

### **Bevacizumab**

As with other solid tumours, including colorectal cancer, breast and lung cancers, therapeutic agents with a specific tumour target are now entering study in gastric and gastroesophageal junction tumours. One of the first compounds studied was Bevacizumab, a humanized monoclonal antibody that binds the vascular endothelial growth factor ligand. In gastric cancer, Shah et al. have reported the results of a phase II to study combining cisplatin plus irinotecan with Bevacizumab. Bevacizumab was given at 15 mg/kg on a once every 3-week basis and concluded that Bevacizumab could safely be given with cytotoxic chemotherapy, including in patients in whom the primary tumour was still in place.

### **Epidermal Growth Factor Receptor Tyrosine Kinase Inhibitors**

Both erlotinib and gefitinib have been studied in gastroesophageal junction and gastric cancers. Cetuximab, an antibody to the epidermal growth factor receptor, is undergoing study as a single agent and also in combination with systemic cytotoxic chemotherapy.

### **Gastric outlet obstruction due to Lymphoma**

Distal subtotal radical gastrectomy is performed if the lesion is present in body or antrum. Spleen is removed at all operations and needle biopsy specimen taken from liver. Following histologic proof, radiation therapy is used as

primary treatment in some centres. Chemotherapy is used when lymph nodes are positive or if there is evidence of systemic disease.

### **Gastric outlet obstruction due to Malignant Smooth Muscle Tumours of Stomach**

Distal subtotal radical gastrectomy is performed for distal lesions and adjuvant CT in metastatic disease with cyclophosphamide, dacarbazine, vincristin and adriamycin.

### **Gastric outlet obstruction due to Duodenal Malignancies**

Pancreaticoduodenectomies are performed when cure is possible. Palliative procedures include duodenoduodenostomy, duodenojejunostomy, gastrojejunostomy and cholecystojejunostomy.

### **Gastric outlet obstruction due to Carcinoma Head of Pancreas**

Whipple's pancreaticoduodenectomy is done if the growth is resectable. Otherwise cholecystojejunostomy to relieve biliary obstruction and gastrojejunostomy to relieve gastric outlet obstruction.

### **Gastric outlet obstruction due to Adult Hypertrophic Pyloric Stenosis**

Finney's or Heineke-Mikulicz pyloroplasty is advised. Ramstedt's Pyloromyotomy has the disadvantage of producing a diverticulum. When diagnosis is in doubt, limited partial gastrectomy with a Billroth I type anastomosis is done.

### **Gastric outlet obstruction due to Willkie's Syndrome**

Conservative treatment is successful in most cases associated with orthopaedic conditions. In chronic SMA syndrome, if the patient fails to respond to conservative treatment (turning to prone or knee-elbow position after meals, prokinetic drugs and weight gain) due duodenojejunostomy is the treatment of choice. Gastrojejunostomy plays no role because of high incidence of stomal ulceration.

### **Gastric outlet obstruction due to Foreign Bodies**

If a foreign body can traverse G-E junction it can pass through the alimentary tract without causing further harm in most cases. Indications for active treatment are

1. Failure to progress
2. Signs of penetration or actual perforation
3. Objects unlikely to move-on
4. Large number of FBs
5. Evident GI haemorrhage

Many of the retained FBs can be removed through OGDscope after they are protected by a sheath. In surgical intervention, gastrotomy or puncture in cases of slender and sharp objects which are closed after a preoperative radiogram, when retrieval of all the FBs is dubious.

**Gastric outlet obstruction due to Bezoars**

Bezoars can be broken endoscopically by laser lithotripsy or mechanically and allowed to pass through. Enzymatic (cellulose) fragmentation is also used. Large trichobezoars need gastrotomy.

**Gastric outlet obstruction due to Crohn's disease**

Resection of the segment if it is short or else bypass is a satisfactory procedure.

**Gastric outlet obstruction due to Gall Stones**

Endoscopic fragmentation or retrieval is advised. If surgical intervention is planned, duodenolithotomy, division of fistula and cholecystectomy is the treatment of choice.

## **MATERIALS AND METHODS**

The patients for this dissertation have been selected from Rajiv Gandhi Govt. General Hospital, Chennai from October 2013 to September 2014. Totally, 50 in-patients of gastric outlet obstruction have been studied.

### **Inclusion Criteria**

1. Patients presenting with gastric outlet obstruction who are treated on inpatient basis.
2. Patients willing for investigations and treatment.

### **Exclusion Criteria**

1. Patients aged 20 years and below.
2. Patient with recent history of any abdominal surgeries.

An elaborate study of these cases with regard to the history, clinical features, routine and special investigations, pre-operative treatment, operative findings, postoperative management and complications in post-operative period is done.

In history, details were noted about presenting complaints, duration, history of acid peptic disease, features of metabolic disturbances, occupation and personal history including diet, bowel and bladder habits, smoking and alcoholism.

Thorough analysis of the findings of physical examination done, which included hydration status, VGP, mass, succussion splash, hepatomegaly and

ascites. Associated conditions like anaemia, hypertension and diabetes were managed before surgery with physician's advice wherever required.

Haemoglobin level, bleeding time, clotting time, routine urine examination, chest screening, ECG, blood grouping, fasting and post prandial blood sugar, blood, urea, serum creatinine, serum electrolytes were estimated as a part of general work-up for surgery. Special investigations like Upper GI Endoscopy, USG abdomen, CECT abdomen were done.

Any one of the following criteria can be used to diagnose gastric outlet obstruction.

- Projectile vomiting of undigested food consumed previous day.
- Palpable hypertrophied stomach
- Visible gastric peristalsis
- Gastric succussion splash 3-4 hours after the last meal
- OGD scopy
- Demonstration at operation of grossly narrowed gastric outlet.

### **Management of cases**

Pre-operative treatment included correction of dehydration, metabolic status, anaemia, IV H<sub>2</sub> blockers; liquid diet and antacids were given along with twice a day stomach wash for a minimum of three days. According to the investigation reports and operative findings, definitive surgery was undertaken.

**Surgeries performed**

- Truncal vagotomy with gastrojejunostomy
- Billroth II gastrectomy
- Billroth II gastrectomy with feeding jejunostomy
- Posterior Gastrojejunostomy
- Total gastrectomy with Roux-en-Y anastomosis
- Anterior gastrojejunostomy alone
- Anterior gastrojejunostomy with limbal anastomosis

**Anaesthesia**

For all cases general anaesthesia was given.

**Post – operative management:**

The patients were managed by Ryle's tube aspiration and Intravenous fluids till the bowel sounds appeared. Oral feeding with fluids was then commenced, solids being given later. Early ambulation was encouraged, especially in elderly patients. Routine antibiotics were given during the immediate post operative period. Regular monitoring of the temperature, pulse, respiratory rate and blood pressure was done.

All the details were recorded in Proforma and master chart is made according to the findings in the Proforma and analysis and discussion carried out and came to a conclusion at the end.

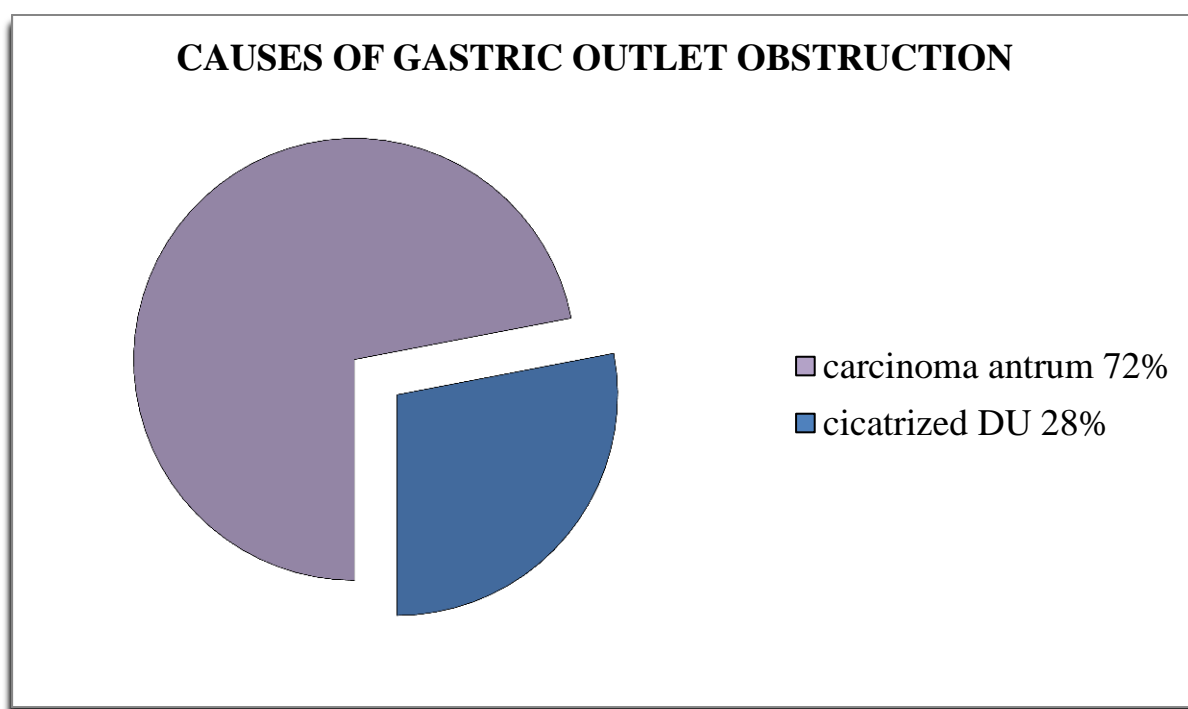


## OBSERVATIONS AND RESULTS

Of the 50 cases of gastric outlet obstruction 36 had carcinoma antrum and 14 had cicatrized duodenal ulcer.

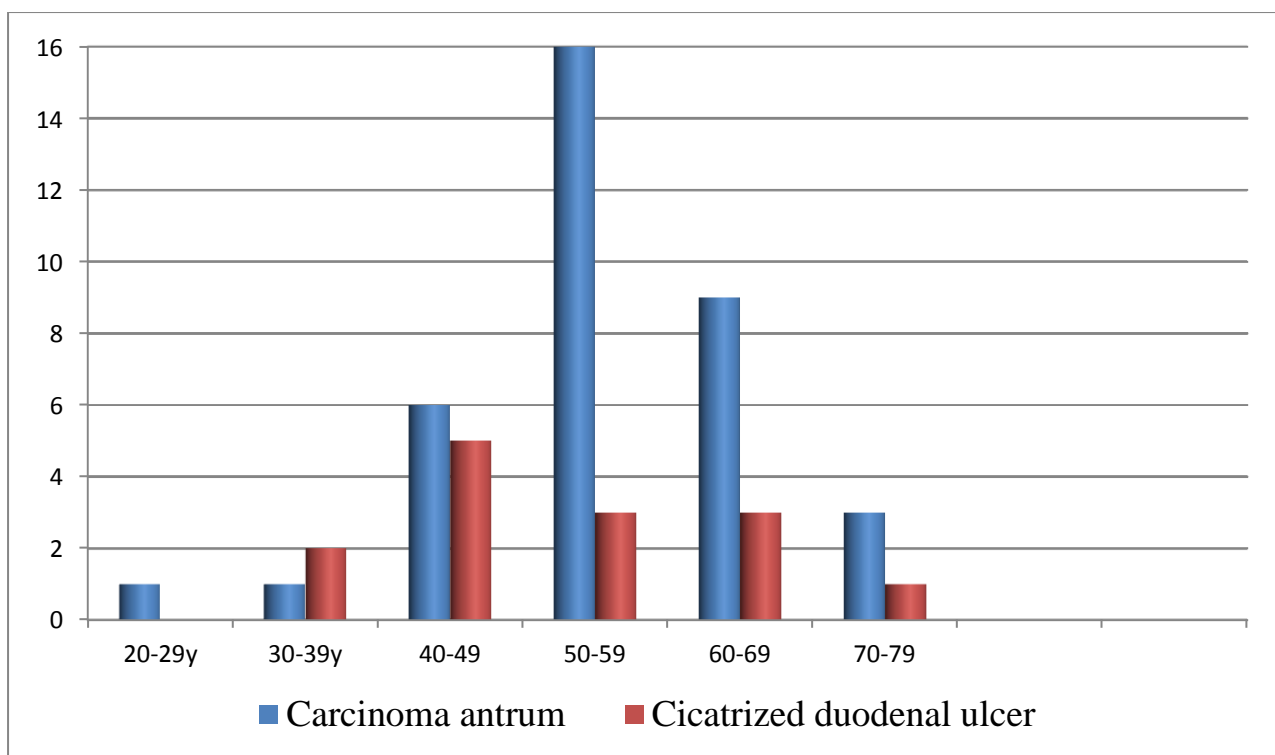
**TABLE 4. CAUSES OF GASTRIC OUTLET OBSTRUCTION.**

Causes	Number of cases	Percentage
Carcinoma antrum	36	72
Cicatrized duodenal ulcer	14	28
Total	50	100



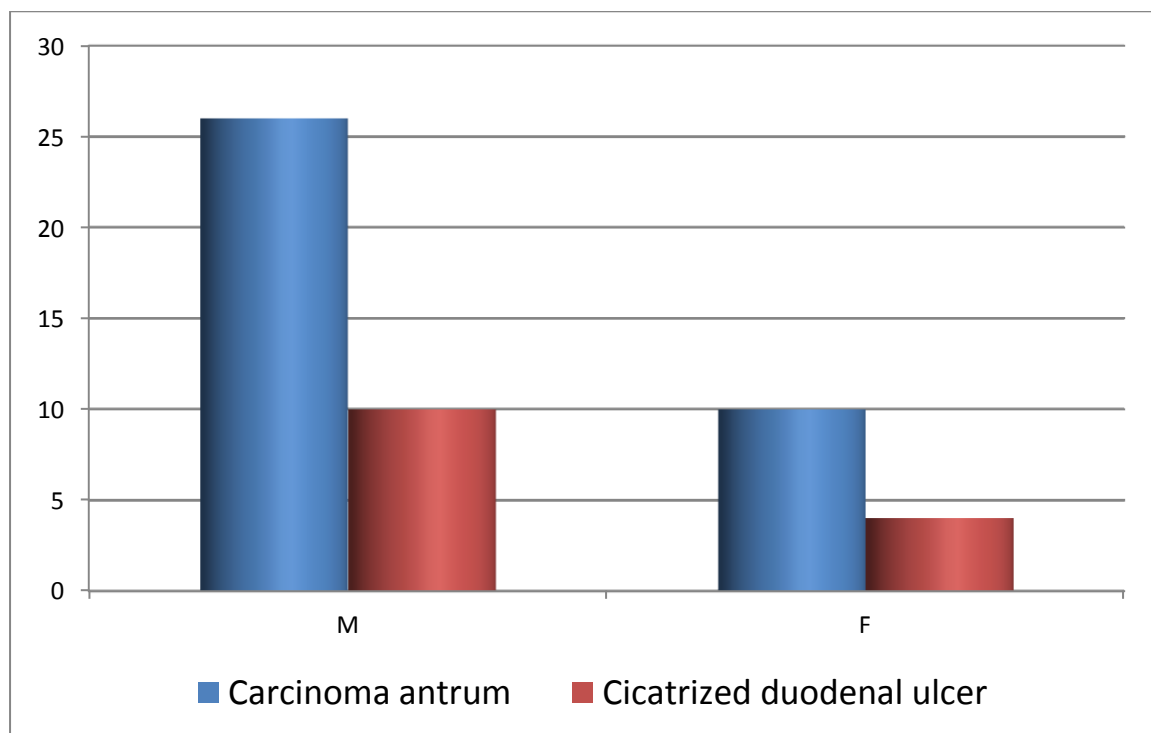
**TABLE 5. AGE DISTRIBUTION.**

Age group	Carcinoma antrum	Cicatrized duodenal ulcer
20-29	1	0
30-39	1	2
40-49	6	5
50-59	16	3
60-69	9	3
70-79	3	1

**AGE DISTRIBUTION.**

**TABLE 6. SEX INCIDENCE**

	Total number of cases	Carcinoma antrum	Cicatrized duodenal ulcer
Male	36	26	10
Female	14	10	4
Total	50	36	14

**SEX INCIDENCE**

## PERSONAL HISTORY IN PRESENT SERIES

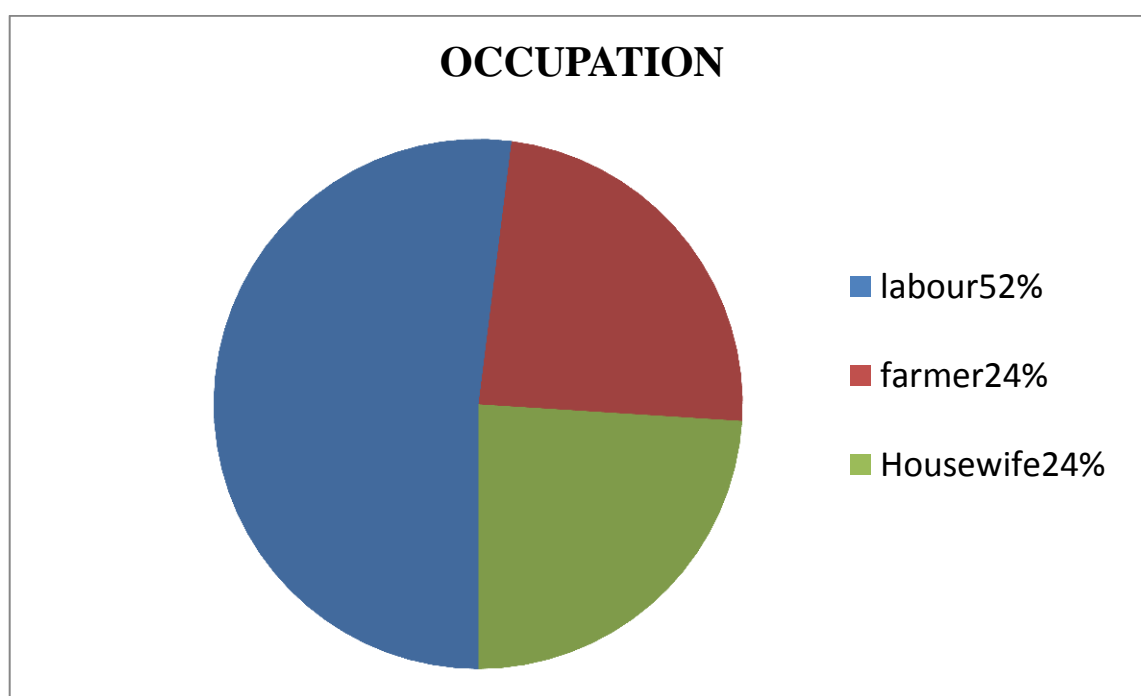
### 1. Socio-Economic Status

Majority of patients were from low socio – economic status.

### 2. Occupation

**TABLE 7. OCCUPATION**

Occupation	Total number of cases	Percentage
Labourer	26	52
Farmers	12	24
Housewife	12	24



### 3. Diet.

90% of patients were taking mixed diet and 10 % patients were taking vegetarian diet. 39 patients (78%) had history of irregular diet habits.

### 4. Smoking.

68% of the patients were smokers in this series and 32% were non – smokers.

### 5. Alcohol

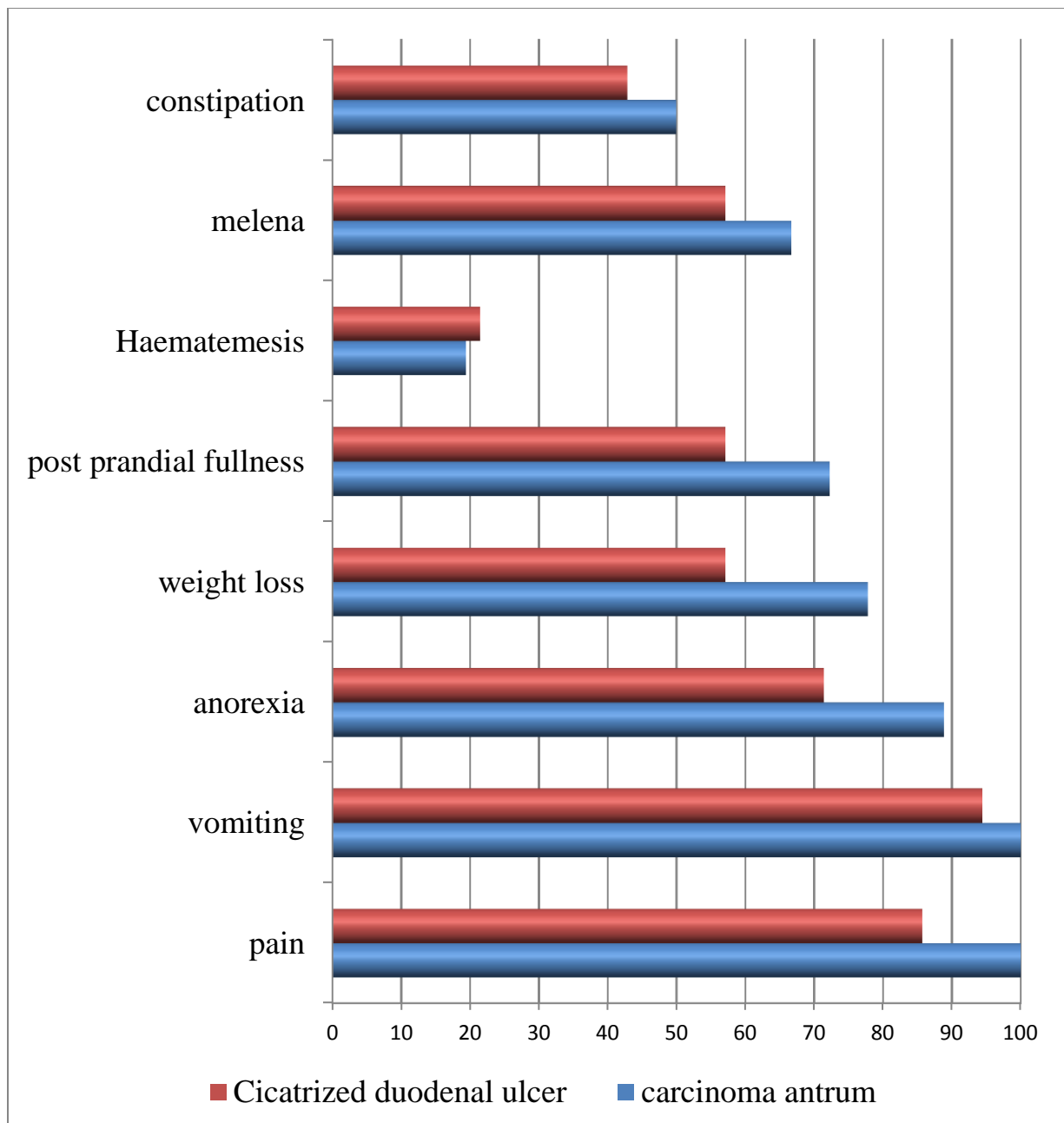
66% of the patients in this series gave history of consuming alcohol.

### SYMPTOMS.

**TABLE 8. SYMPTOMS OF THE PATIENTS PRESENTING AS GOO.**

Symptoms	Carcinoma antrum	Cicatrized duodenal ulcer	Total number of cases
Pain	36(100%)	12(85.71%)	48(96%)
Vomiting	34(94.44%)	14(100%)	48(96%)
Anorexia	32(88.89%)	10(71.43%)	42(84%)
Weight loss	28(77.78%)	8(57.14%)	36(72%)
Post prandial fullness	26(72.22%)	8(57.14%)	34(68%)

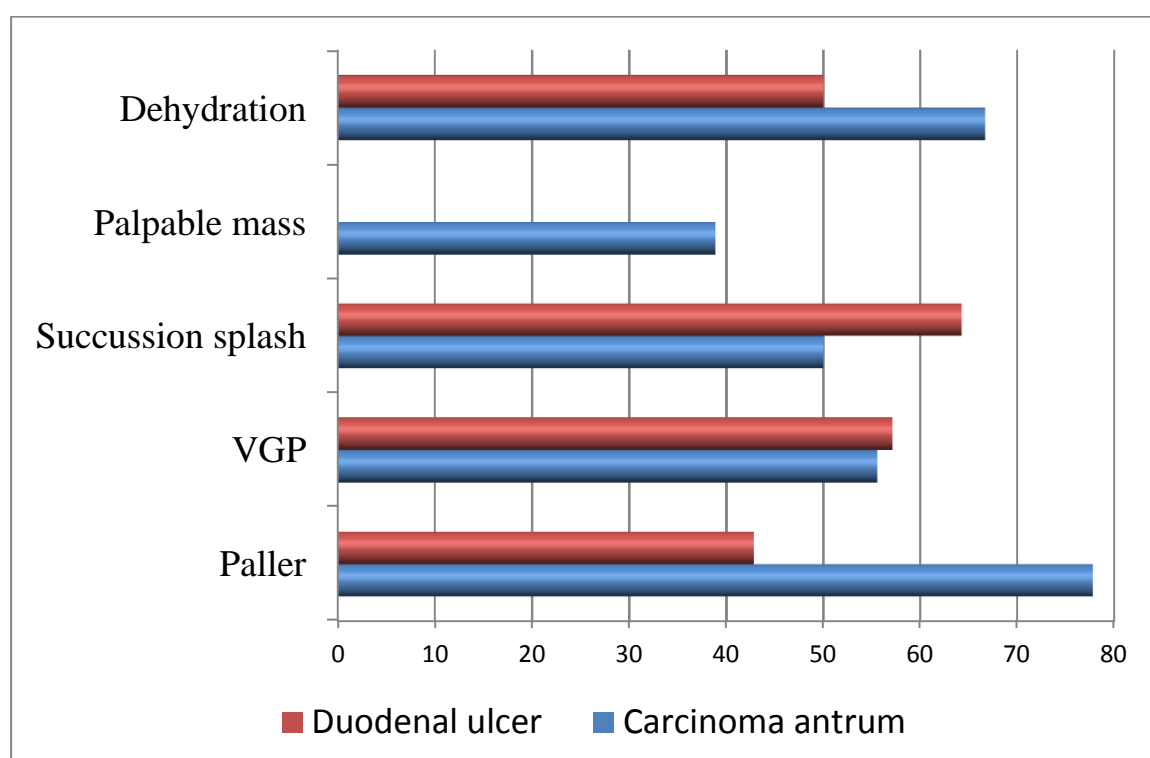
Haematemesis	9(25%)	3(21.43%)	12(24%)
Melena	24(66.67%)	8(57.14%)	32(64%)
Constipation	18(50%)	6(42.86%)	24(48%)



## SIGNS

**TABLE 9. SIGNS IN THE PATIENTS PRESENTING WITH GOO.**

Signs	Duodenal ulcer (%)	Carcinoma antrum (%)	Total number of cases (%)
Pallor	6(42.86%)	28(77.78%)	34(68%)
VGP	8(57.14%)	20(55.56%)	28(56%)
Succussion Splash	9(64.28%)	18(50%)	27(54%)
Palpable mass	0	14(38.89%)	14(28%)
Dehydration	7(50%)	24(66.67%)	31(62%)



## INVESTIGATIONS

The following investigations were carried out before subjecting the patient for surgery. Hb%, FBS, blood grouping, serum electrolytes, urine routine, chest X-ray, ECG, endoscopy, ultrasonography and CECT abdomen done.

## DISTRIBUTION OF BLOOD GROUP

**TABLE 10. DISTRIBUTION OF BLOOD GROUP IN DUODENAL ULCER PATIENTS.**

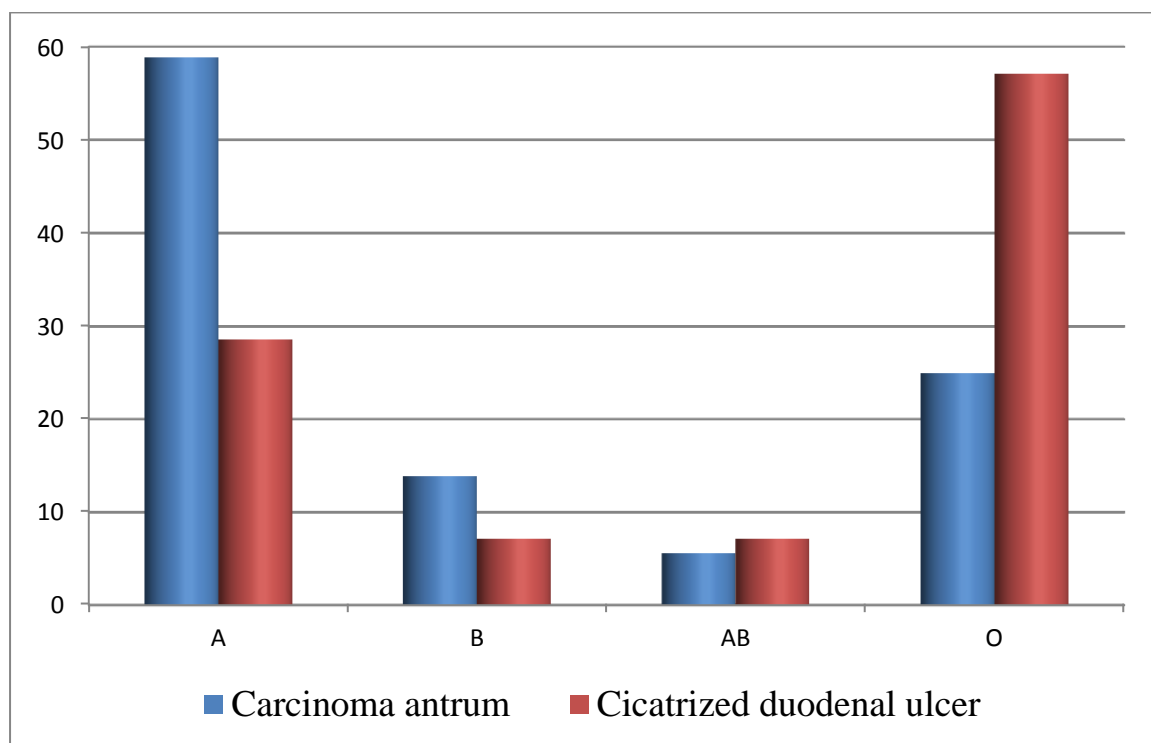
Blood group	Total number of cases	Percentage
A	4	28.57%
B	1	7.14%
AB	1	7.14%
O	8	57.14%

**TABLE 11. DISTRIBUTION OF BLOOD GROUP IN CASES WITH CARCINOMA PYLORIC REGION.**

Blood group	Total number of cases	Percentage
A	20	58.82%
B	5	13.89%



AB	2	5.56%
O	9	25%



### Upper GI Endoscopy

Done in all cases. 36 cases of pyloric carcinoma diagnosed and confirmed with biopsy. 14 had cicatrized duodenal ulcer.

### Ultrasonographic examination

It is done in all cases. Carcinoma pyloric region with ascitis was present in eleven cases. Ascitis with liver secondaries was present in seven case. The rest showed normal study.

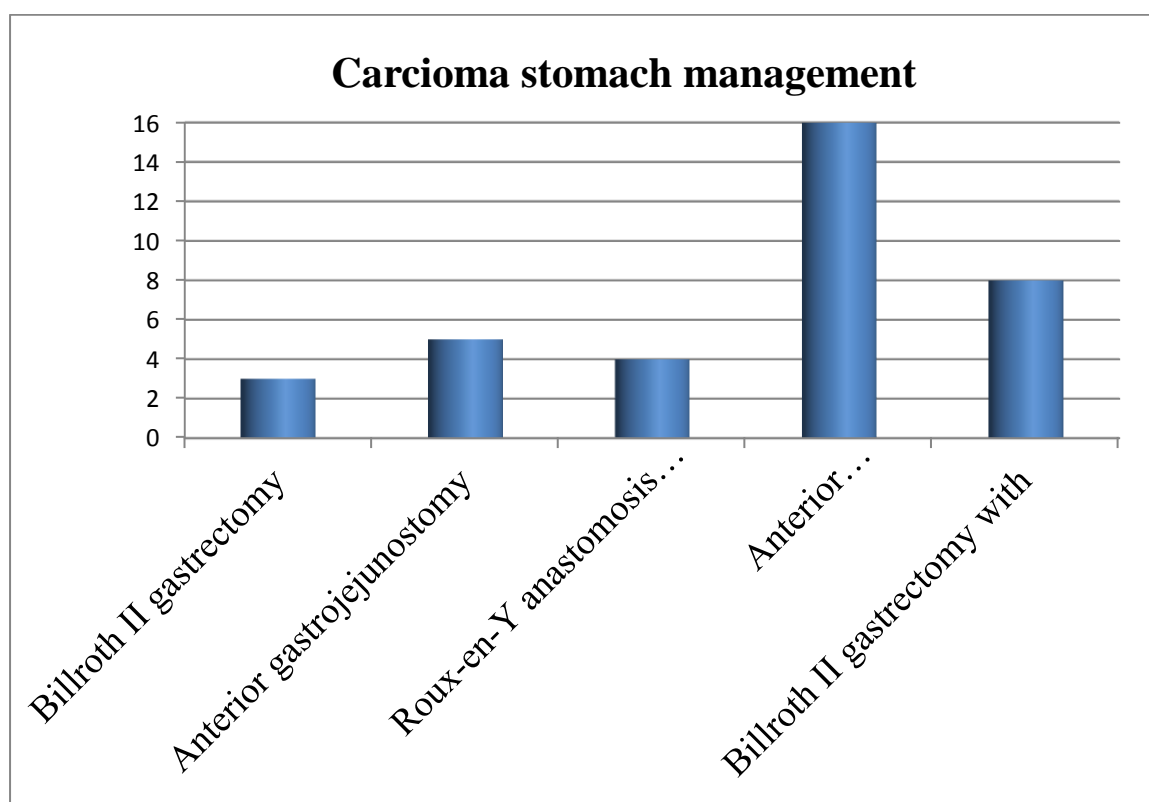
## CECT Abdomen

It is done in all cases. Ten patients have lost the posterior wall fat plane. Fourteen patients have lymphnode enlargement. Eleven patients have ascites. Seven patients have ascites with secondaries liver.

## Serum electrolytes

In present series, all patients were subjected to serum electrolyte estimation, out of them 9 patients showed electrolyte imbalance. All patients underwent pre-operative treatment to get the optimum metabolic status. The pre-operative treatment included liquid diet, liquid antacid and intravenous ranitidine. Stomach wash using no 16 Ryle's tube with normal saline was given twice a day for three days prior surgery.

## TYPES OF SURGICAL PROCEDURES ADOPTED IN THE STUDY



**TABLE 12. THE TYPES OF SURGICAL PROCEDURES  
UNDERWENT BY THE PATIENTS.**

Procedure	Number of cases	Percentage
1. Duodenal ulcer cases		
➤ Truncal vagotomy with Gastrojejunostomy	14	100%
2. Carcinoma antrum cases		
➤ Billroth II gastrectomy	3	8.33%
➤ Anterior gastrojejunostomy	5	13.89%
➤ Roux-en-Y anastomosis after total gastrectomy	4	11.11%
➤ Anterior gastrojejunostomy with limbal anastomosis	16	44.44%
➤ Billroth II gastrectomy with feeding jejunostomy	8	22.22%

**Post operative management**

All the patients were kept nil orally and on Ryle's tube aspiration for duration varying from 3 to 10 days. Oral sips were allowed after removal of Ryle's tube. IV fluids were stopped on the 5th to 10th post-operative day and patients started on semisolid diet. The patients were put on broad spectrum antibiotics, intravenous Protan pump inhibitor and analgesics.

**Post operative complications**

Wound infection developed in two patients who were treated by repeated dressing and appropriate antibiotics. In four patients respiratory tract infection developed which was treated by chest physiotherapy and review of antibiotics. 25 patients of antral carcinoma were treated postoperatively by chemotherapy with 5-fluoro uracil. Rest of the patients had an uneventful post-operative period. Post operative hospitalisation ranged from 7 to 40 days with an average of 11 days.

## DISCUSSION AND ANALYSIS

The discussion is mainly on analysis and observation made regarding the presenting symptoms, signs, investigations, operative findings, management and postoperative events in 50 cases of gastric outlet obstruction admitted to Rajiv Gandhi Government General Hospital during the period October 2013 to September 2014.

Of 50 cases,

Gastric outlet obstruction secondary to carcinoma pyloric region – 36

Gastric outlet obstruction secondary to cicatrized duodenal ulcer – 14

The commonest cause of gastric outlet obstruction is carcinoma of pyloric antrum. The next commonest cause is cicatrized duodenal ulcer. These observations reveal that the incidence of gastric outlet obstruction secondary to chronic duodenal ulcer has come down while that of malignancy has relatively increased.

In this study most patients were in the fifth decade. In chronic duodenal ulcer cases the maximum incidence seen in the age group of 40-49 years. The average age being 52.35 years with a span from 35 to 76 years. Men outnumbered women by 3.7:1. In the series of Fisher et al., the average age was 54 with a span from 20 – 89 years and men outnumbered women by 2:1.

In antral carcinoma cases, the maximum incidence is seen in the age group of 50-59 years. The youngest age of presentation is 29 years and the oldest is 72 years with the average being 56.67 years. Men outnumbered women

by 2.6:1 as compared to 5.5:1 observed by Yogiram and Chowdhary. This higher incidence in males, worldwide can be explained as because of more consumption of gastric irritants by males compared to females.

44% of the patients were manual labourers who gave a history of irregular diet habits, which seemed to contribute to disease process. The series of Donald D. Kozoll and Karl A. Meyer also showed the same pattern with the non-skilled day labourer group listed most frequently with obstruction.

In this series 68% of patients had history of smoking and 66% had history of alcohol intake. Donald D. Kozoll and Karl A. Meyer reported this to be 76.2 and 52.3% respectively. This points to the commonly observed fact that a higher incidence of use of alcohol and tobacco is seen in these patients and are significant risk factors.

Post – prandial vomiting and epigastric pain are the main symptoms (96%) in this series. Vomiting is usually spontaneous and projectile type containing partially digested food particles. Other symptoms included anorexia (84%), weight loss (72%), post prandial epigastric fullness (68%), haematemesis (24%), malena (64%) and constipation (48%). In the series of Michael L. Schwartz et al.,[76] post prandial vomiting was the commonest symptom (91%). Other symptoms included epigastric pain (86%) and weight loss (52%).

In the series of Yogiram and Chowdhary epigastric pain was the commonest symptom (87%). Other symptoms included post – prandial vomiting

(80%) and constipation (30%). Keith A. Kelly in his series, reported intractable vomiting and weight loss in 54% of patients and upper gastro intestinal haemorrhage in 34%. Weight loss was seen in 59.5% of patients in the series of Donald D. Kozoll and Karl A. Meyer and 32% in the series of Harvey J. Dworken and Harold P. Roth. Thus weight loss seemed to be significant in patients with pyloric obstruction and this points to the long standing nature of the disease and the need for proper pre operative nutritional supplementation in these patients.

In carcinoma pyloric antrum cases, pain (100%) was the leading symptom. Other symptoms included vomiting (94.44%), anorexia (88.89%), weight loss (77.78%), post prandial fullness (72.22%), Haematemesis was present in (25%), malena in (66.67%) and constipation in 50%.

Pain, vomiting, anorexia and post prandial fullness (100%) were the leading symptoms in gastric outlet obstruction due to cicatrized duodenal ulcer.

Pallor was present in 77.78% and dehydration in 66.67%. In the series of Michael L. Schwartz et al., dehydration was present in 22%.

Visible gastric peristalsis was seen in 64.28% of cicatrized duodenal ulcer cases. In the series of Yogiram and Chowdhary visible gastric peristalsis was present in 74%.

Succussion splash was seen in 64.28% of cicatrized duodenal ulcer cases while Harold Ellis observed succussion splash in 64% of his cases.

Visible gastric peristalsis (55.56%) and succussion splash (50%) were less prominent in malignant cases. This corresponds in observation made by Harold Ellis. Palpable mass was present in 38.89% of malignant cases.

Blood group 'O' was common in cicatrized duodenal ulcer patients (57.14%) followed by blood group 'A' (28.57%). This is significant as persons of blood group 'O' are about three times more likely to develop acid peptic disease than persons of other blood groups. Blood group 'A' was common in malignant cases (58.82%).

In the present series, 100% of cicatrized duodenal ulcer patients underwent truncal vagotomy with gastrojejunostomy.

In carcinoma antrum cases, 3 patients (8.33%) underwent Billroth II Polya gastrectomy and 5 patients (13.89%) underwent anterior gastrojejunostomy. 4 cases (11.11%) underwent Roux-en-Y anastomosis after total gastrectomy while 16 cases (44.44%) underwent Anterior gastrojejunostomy with limbal anastomosis. The remaining 8 cases (22.22%) underwent Billroth II gastrectomy with feeding jejunostomy.

All the patients were subjected to a standard pre-operative treatment, which included stomach wash twice a day for three days prior to surgery. Pre-operatively stomach was dilated in majority of the cases. Post-operatively aspiration was continued till bowel movements established by noting bowel sounds, passing of flatus and gross reduction in quantity of Ryle's tube



aspiration. Later on patients were allowed to take oral fluids and then liquid and solid diet.

In this series two patients had wound infection and were treated by repeated dressing and appropriate antibiotics. Four patients had respiratory tract infection and were treated by review of antibiotics and chest physiotherapy. Mortality rate was zero in this study.

15 patients of antral carcinoma were treated postoperatively by chemotherapy with 5-fluoro uracil.

Most of the stenosing duodenal ulcer cases have no recurrence of symptoms in any of the cases that turned up for follow up.

## CONCLUSION

Since the study has been based on a small number of cases, with a limited follow up, it is rather difficult to come to definite conclusions. However some of the conclusions which can be drawn from this series are as follows:

1. The commonest causes of gastric outlet obstruction in adults are carcinoma stomach with antral growth producing gastric outlet obstruction (72%) and cicatrised duodenal ulcer (28%).
2. In the vast majority of cases, the diagnosis can be established clinically.
3. Upper Gastro intestinal endoscopy should be mandatory in all suspected cases of gastric outlet obstruction. It can diagnose the cause of obstruction very effectively than any other investigative modality.
4. Number of cases with cicatrised duodenal ulcer as the chief etiological factor for gastric outlet obstruction is diminishing and the number of cases of antral carcinoma of stomach as the cause of gastric outlet obstruction is increasing.
5. Effective treatment in carcinoma stomach depends on early diagnosis.

## SUMMARY

The findings of this study include

1. The most common cause of gastric outlet obstruction is carcinoma stomach with antral growth producing gastric outlet obstruction (72%) in this study.
2. Number of cicatrised duodenal ulcer producing gastric outlet obstruction was 28% in this study.
3. Males are more commonly affected than females and the male – female ratio is 2.6:1 in malignancy.
5. This study was undertaken in an adult population.
6. The most common presenting complaints were vomiting (96%), abdominal pain (96%) and loss of appetite (84%). Loss of weight (72%) was also a common complaint.
7. Visible gastric peristalsis and succussion splash were less prominent in malignant cases when compared to stenosing duodenal ulcer cases.
8. 39% of the malignant cases (14 cases) presented with mass in the upper abdomen.
9. 15 cases of malignancy were able to undergo definite surgical procedure. Others were all underwent palliative procedures.
10. The surgical procedure undertook in all the cicatrised duodenal ulcer patients were truncal vagotomy and posterior gastro jejunostomy and there were no recurrence of symptoms in any of the cases which turned up for follow up.

The incidence of gastric outlet obstruction secondary to cicatrised duodenal ulcer has reduced probably due to (a) Increased awareness of the disease (b) Change in dietary habits and (c) availability of drugs like H<sub>2</sub> receptor blockers, proton pump inhibitors and effective antibiotics which can eradicate *Helicobacter pylori* .

The availability of flexible fibre – optic endoscope has helped the surgeon to know the cause of obstruction (especially in malignancy) pre-operative and biopsy taken from the suspected lesion can confirm the diagnosis also.

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## **PROFORMA**

### **I. PARTICULARS OF THE PATIENT:**

IP No:

Name:

Age:

Sex:

Address:

Socio - economic status:

Date of Admission:

Date of Discharge:

Final Diagnosis:

### **II. COMPLAINTS WITH DURATION:**

- Abdominal Pain:

- Vomiting:

- Loss of weight:

- Loss of Appetite:

- Constipation:

- Other Complaints:

### **III. DETAILS OF PRESENTING COMPLAINTS:**

- Abdominal Pain: - Duration, Site, Nature, Relation to food, Radiation,

Aggravating and relieving factors

- Vomiting: - Duration, Frequency, Quantity, Relation to food, Induced / Spontaneous, Forcible / not forcible, Foul smelling, frothy, Digested / undigested contents, Bile / blood / mucus,
- Details of other complaints:

#### **IV. PAST HISTORY:**

- History of peptic ulcer disease:
- History of swallowing corrosive substances:
- Any other relevant history:

#### **V. PERSONAL HABITS**

- Smoking: duration and quantity
- Alcohol intake: duration, quantity and frequency
- Irregular meals
- Irritating food, inadequate mastication:

#### **VI. FAMILY HISTORY**

- Any history of relevance:

#### **VII. GENERAL PHYSICAL EXAMINATION:**

- Built: thin / muscular / fat
- Loss of skin elasticity:
- Dehydration: mild / moderate / severe
- Vitamin deficiencies:
- Pallor:
- Jaundice:

- Lymph nodes:

**Systemic Examination:**

- Cardio vascular system:
- Pulse rate, volume.
- B.P:
- Precordium:
- Respiratory System:
- Central Nervous System: Tetany:

**VIII. ABDOMINAL EXAMINATION:****Inspection:**

- Abdomen retracted / distended:
- Umbilicus:
- Visible veins:
- Visible peristalsis, location, direction and how included:
- movement with respiration :
- other signs:

**Palpation:**

- Site of tenderness:
- Mass
- Tenderness over mass
- Site, Size, Shape, Surface:
- Consistency:

- Mobility - mobile / restricted / fixed
- Movement with respiration

**Pulsation / transmitted pulsations:**

- succussion splash :
- Liver and spleen: Palpable or not
- Spine, renal angles:
- External genitals:
- Shifting dullness / fluid thrill:
- ausculto - percussion of stomach : size and shape :
- Other findings:

Rectal and vaginal examinations:

**IX. INVESTIGATIONS:**

Routine investigations: Blood :

- Hb,
- ESR: mm/hr:
- WBC TC, DC.
- Blood urea:
- Serum Creatinine:
- Blood group:
- Serum Electrolytes: Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup>, HCO<sub>3</sub><sup>-</sup>

Urinalysis: Albumin, Sugar, Microscopy& pH,

Stool examination for ova, cysts and occult blood:



Chest X-ray and E.C.G:

**Specific investigations :**

- Upper G.I. Endoscopy:

-CECT Abdomen:

**X. DIAGNOSIS:**

**XI. TREATMENT:**

Pre-operative:

- Correction of water and electrolyte balance:

- Correction of anaemia:

- Stomach washes:

**Operative:**

Laparotomy findings and procedure performed:

Post - operative course:

Histo - pathology report (if any):

**XII. CONDITION AT DISCHARGE:**

**XII. FOLLOW -UP:**

**XIV. COMMENTS AND SUMMARY:**

**ABBREVIATION TO MASTER CHART**

A -Anorexia

AB -Abnormal

AGJ -Anterior Gastro jejunostomy

ASC -Ascites

B -Burning

BK -Back

BII -Billroth II gastrectomy

C -Constipation

Che -Chemotherapy

CaPyl -Carcinoma pyloric region

CzDU -Cicatrized Duodenal Ulcer

DOA -Date of Admission

DOS -Date Of Surgery

DOD -Date Of Discharge

d -days

DA -Dull aching

Epi -Epigastric

Fa -Farmer

F -Food

Fi -Fixed

H -Haemetemesis

HW -Housewife

H -Hard

Ir -Irregular

L -Labourer

L+ -Liver Metastasis

LA -Limbal Anastomosis

LN -Lymph node enlargement

LOFP -Loss of fat plane.

LRTI -Lower respiratory tract infection

m -months

Med -medicine

M -malena

Mi -Mixed

NOR -Normal

PPF -post prandial fullness

S -smooth

TV -Truncal Vagotomy

V -vomiting

Veg -Vegetarian

WI -wound infection

Y -years